Atrial arrhythmias after lung transplant: A call to action starting with the electrocardiogram

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Atrial flutter after lung transplantation.

Central Message

If we are to have an impact on outcomes when arrhythmias complicate lung transplantation (or any surgical procedure), we must make a precise diagnosis of the arrhythmia.

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In this issue of the *Journal*, D'Angelo and colleagues¹ published their evaluation of the impact of atrial arrhythmias (AAs) occurring in the first month after lung transplantation in adults. The survival over the ensuing 5 years was significantly worse in those with early AAs than in those who maintained normal sinus rhythm. This was noted despite the fact that all those with AAs ultimately converted back to normal sinus rhythm ("confirmed" by physical examination) before discharge from the hospital. The arrhythmias were presumably atrial flutter and atrial fibrillation. Advanced age and prior coronary artery bypass surgery were risk factors for the development of AAs. A history of AAs was protective against post-transplant arrhythmias, possibly the consequence of treatment of the AAs. There are multiple questions raised by this study. (1) What are the AAs specifically? (2) Why do AAs occur after lung transplantation? (3) Why is the survival worse for those with post-transplant AAs? (4) What can be done to prevent the arrhythmias? (5) What should be done for treatment? In any analysis of AAs, the answer to question 1 is imperative because it affects the subsequent approach to evaluation and management. Despite detailed definitions of AAs in the Society of Thoracic Surgeons data dictionary, this study did not differentiate between them, the justification being historical precedent. It is time to channel the power of the Society of Thoracic Surgeons and adopt similar precision.

Atrial fibrillation is relatively common after a variety of major operations, particularly in elderly adults with an incidence of 30% after coronary artery bypass surgery,² 20% after thoracotomy with lobectomy,³ and 12% after open repair of an abdominal aortic aneurysm.⁴ The precise reason for atrial fibrillation after any of these has not been determined. Measures to prevent atrial fibrillation, particularly after coronary artery bypass surgery, have been studied extensively with a variety of randomized studies without a clear consensus. The technique for the pulmonary venous anastomosis in lung transplantation involves excision of the orifices of the pulmonary veins

along with the intervening atrial tissue on each side and sewing the pulmonary venous cuff to the remaining lateral atrial wall; this is the ultimate form of pulmonary vein isolation, a relatively common procedure performed for the treatment of atrial fibrillation.⁵ A study by Gandhi and colleagues,⁶ of an animal model of the atrial incisions associated with lung transplantation, found that these incisions actually provide the substrate for atrial flutter, the mechanism being a reentrant circuit around the area of conduction block produced by the atrial suture lines for the pulmonary venous anastomoses. Further, they noted that the conduction from the left atrium to the right atrium was not consistently 1:1, producing irregular R-R intervals mimicking atrial fibrillation. This "left atrium flutter" results in atypical "flutter" waves on the surface electrocardiogram (Figure 1). It is attractive to ascribe the AAs after lung transplantation to this mechanism because the "cure" for it can be obtained with radiofrequency catheter ablation in the electrophysiology laboratory.

Why is the long-term survival affected by AAs occurring in the early post-transplant period? In general, people do not die of lone atrial fibrillation, but rather as a result of the associated cardiac comorbidities.⁷ In this study,¹ echocardiographic measures such as left atrium size and volume and left ventricular ejection fraction were not collected or reported, and cardiac catheterization was performed more than 1 year before transplant. Thus, no assessment of myocardial substrate is available to evaluate the proper context for these AAs. The causes of death in the group reported by D'Angelo and colleagues¹ are not

FIGURE 1. This is a 12-lead electrocardiogram from a patient in atrial flutter after lung transplantation. This has an atypical pattern of “p” waves compared with the more typical electrocardiogram in atrial flutter.

provided. The most likely explanation must be that the development of AAs serves as a marker of underlying cardiovascular risk. However, given the limitations of this analysis, one cannot exclude the possibility that the treatment of the AAs or some other unidentified factor accounts for the risk. It should be noted that other studies of AAs after lung transplantation have not shown a reduced survival.⁸ How could treatment for AAs reduce survival? One would think that efforts to maintain sinus rhythm would enhance survival of individuals with AA. Antiarrhythmic drugs, even digoxin,⁹ are known associates with increased mortality in other populations. There is also a case report of amiodarone toxicity in a patient with AAs after lung transplantation.¹⁰

The bottom line from the study by D’Angelo and colleagues¹ is that the development of AAs after lung transplant is associated with a longer intensive care unit stay, longer hospital stay, and worse survival. Why the survival is worse cannot be adequately addressed in the current analysis. Pretreatment in those at risk (age >60 years and history of coronary artery disease) may blunt this effect. As emphasized, any future evaluation mandates specificity of rhythm diagnosis and rigorous collection of objective data and known cardiac associations. Normal sinus rhythm is a diagnosis best made by electrocardiogram and not physical examination.

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