Iatrogenic pleuropericardial communication: A rare complication of percutaneous epicardial mapping

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Introduction
Ablation of ventricular tachycardia (VT) is a well-established treatment for patients with recurrent implantable cardioverter-defibrillator shocks. Epicardial access is increasingly performed when epicardial arrhythmogenic substrate is suspected and may be considered as a first-line approach in the subset of myocardial diseases with preferential epicardial substrate, namely, idiopathic dilated cardiomyopathy, arrhythmogenic right ventricular dysplasia, or chagasic cardiomyopathy. Cardiac complications of this approach include right ventricular puncture, pericardial bleeding, coronary vessel damage, and pericarditis. Damage to extracardiac structures may occur during puncture, mapping, and ablation; deep knowledge of the anatomic relationships of the heart and prompt recognition of complications are paramount to minimize procedural risk. We report the first case of acute iatrogenic pleuropericardial communication during epicardial mapping in a patient with chagasic cardiomyopathy.

Case report
We present the case of a 66-year-old woman with a history of chagasic cardiomyopathy, severe left ventricular systolic dysfunction, and an implanted single-chamber implantable cardioverter-defibrillator due to prior VT. The patient was scheduled for a combined endo-epicardial substrate-based ablation procedure because of recurrent, poorly tolerated VT despite antiarrhythmic treatment (amiodarone 200 mg/d) with electrocardiographic criteria for epicardial origin (Figure 1A). The baseline chest radiograph showed absence of pleural effusion (Figure 1B).

After uneventful subxiphoid epicardial access a 3.5-mm irrigated-tip ablation catheter (SmartTouch, Biosense Webster Inc., Diamond Bar, CA) was advanced through a steerable sheath (Agilis EPI, St. Jude Medical, Inc., St. Paul, MN). The catheter moved freely in the pericardial space during mapping, recording extensive areas of dense scar at anterior and inferolateral walls, with presence of multiple late potentials. During mapping, inadvertent passage of the catheter to an extracardiac structure was observed, extending lateral and anterior, where electrical silence was recorded. The site of communication was observed in the most inferoposterior area of the left ventricle, where a clear breakthrough between the pericardial and pleural space was mapped and high contact force was observed (Figure 2 and Online Supplemental Video). The catheter was then relocated in the pericardial space to complete mapping and ablation. Intraprocedural transesophageal echocardiography was performed because of inability to drain saline through the epicardial sheath, revealing absence of pericardial but significant pleural effusion (Figure 2). Epicardial ablation was completed within 26 minutes of radiofrequency delivery. After the ablation catheter was removed, drainage of pleural effusion was achieved through the subxiphoid steerable sheath located in close proximity to the communication site (700 cm³ of clear saline without hematic remnants). Endocardial ablation was also performed through transseptal access to complete substrate modification. The postprocedural chest radiograph revealed a mild residual left-sided pleural effusion (Figure 2), and the patient did not complain of any symptoms related to the procedure. A repeat endo-epicardial ablation procedure was performed 2 weeks later because of VT recurrence without any evidence of pleural effusion during and after the procedure, suggesting resolution of the pleuropericardial communication.

Discussion
Subxiphoid epicardial access is increasingly used in the electrophysiology laboratories to ablate a variety of substrates, mainly VT. Deep knowledge of the anatomy of the epicardial space, including both cardiac and extracardiac structures, as well as the potential complications of this approach, is crucial to safely perform epicardial procedures. We report a case of inadvertent passage of an ablation catheter from the pericardial to pleural space, probably
because of high contact force applied toward the pericardium. Prompt recognition of this event is important to avoid roving the catheter and ablate in the pleural space to avoid the risk of pneumothorax, hemothorax, or phrenic nerve injury.

Congenital pleuropericardial fistula is a rare condition that may constitute an alternative explanation to the communication of both anatomical spaces in this patient. During the repeat procedure, saline accumulated in the pericardial space and no pleural effusion was observed, suggesting resolution of the communication and reasonably ruling out this preexisting condition. However, contrast injection into the pericardial space was not performed during the index or repeat procedure, which could have provided some evidence for a preexisting communication, presence of adhesions, and confirmation of resolution of the fistula.

Chagasic cardiomyopathy is a parasitic inflammatory condition affecting the myocardium and pericardium during the acute presentation and the myocardium at its chronic phase, potentially leading to a postinflammatory, friable pericardium prone to perforation if high contact force is applied. Likewise, pericardial lesions due to radiofrequency ablation were reported to cause iatrogenic pleuropericardial fistula, probably through inflammation. However, the lack of pericardial adhesions during the index procedure makes this mechanism unlikely.

**Conclusion**
This is the first report of an iatrogenic pleuropericardial communication complicating an epicardial VT ablation procedure. High contact force should be avoided in the

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**Figure 1**  
A: Electrocardiogram of the clinical ventricular tachycardia. B: Preprocedural chest radiograph.
pericardial space when catheter is directed toward the pericardium.

Appendix
Supplementary data

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.hrcr.2016.08.010.

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