Bilateral Large Pneumothoraxes Following Implantable Cardioverter-Defibrillator Generator Change: A Case Report of an Uncommon Event Complicating a Common Procedure

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ABSTRACT

Introduction: A bilateral large spontaneous pneumothorax to our knowledge has never been reported after a device implantation. We report an unusual case of a patient developing spontaneous bilateral large pneumothoraxes after an implantable cardioverter-defibrillator generator and lead revision without evidence of any obvious traumatic cardiac injury.

Case Presentation: A 79-year-old white man was scheduled for implantable cardioverter-defibrillator generator change and addition of an atrial lead. Approximately one hour after the procedure, he suddenly went into respiratory distress with profuse sweating, and pallor with falling oxygen saturation and blood pressure. Chest x-ray showed bilateral large pneumothoraxes.

Discussion: In our literature search, we found no reports of large bilateral pneumothorax in the absence of any traumatic cardiac or lung injury. Rupture of bilateral pleura during subclavian access or presence of pleuropleural communication or a right atrial microperforation could be possible causes.

INTRODUCTION

Pneumothorax following device implantation through subclavian venous access is uncommon and occurs during 1% to 2% of procedures with experienced operators. Most commonly, pneumothorax occurs on the ipsilateral side and is associated with venous puncture. Pneumothorax may be detected during a procedure or within 24 hours of implantation. Unlike ipsilateral pneumothorax, contralateral pneumothorax may be caused by perforation of the cardiac wall, pericardium, and pleura. We know of only one reported case of bilateral pneumothoraxes following device implantation with no apparent cause. In the literature, bilateral spontaneous pneumothoraces have been documented as “buffalo chest syndrome” with the cause hypothesized as congenital pleuropleural communication. In all cases, the pneumothorax was reported several days after the procedure. We conducted an extensive literature review using Google Scholar, Ovid, and MEDLINE/PubMed. The search included the MeSH terms pneumothorax, bilateral pneumothorax, pacemaker implantation, subclavian puncture, pericardial effusion, cardiac trauma, and pleural injury and dated back to January 1, 1975. To our knowledge, no cases of large bilateral spontaneous pneumothoraces have been reported. In this report, we describe the trajectory of a patient who developed spontaneous bilateral pneumothoraxes following an implantable cardioverter-defibrillator (ICD) generator change with atrial lead insertion and no direct evidence of traumatic cardiac injury.

CASE PRESENTATION

A 79-year-old white man with a history of coronary artery bypass graft surgery, severe ischemic cardiomyopathy (left ventricular ejection fraction, 30% to 35%), hypertension, diabetes mellitus, and paroxysmal atrial tachyarrhythmia was scheduled for an ICD generator change and addition of an atrial lead. His original ICD was implanted in 2005, with a single ventricular lead. The patient was a former smoker and had no known pulmonary history. He underwent an uneventful generator change and atrial lead placement through left subclavian access. Both atrial lead and existing right ventricular lead measurements were noted as normal before closure. There was a transient drop in blood pressure during the procedure from 130/90 mmHg to 100/70 mmHg. To rule out possible cardiac injury, an echocardiogram was performed in the laboratory before recovery, and there was no evidence of pericardial effusion.
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The ICD generator was a Medtronic Evera XT DR Model DDBB1D1 (Medtronic, Inc, Minneapolis, MN) with a pacing mode AAIR with a default to DDDR if needed at a lower pacing rate of 60 beats per minute. The newly inserted atrial lead was a Medtronic active fixation Model 5076-45 cm ([Medtronic, Inc, Minneapolis, MN] right atrial P-wave 2.3 mV, right atrial pacing threshold 1.6 V at 0.5 milliseconds, and lead impedance 1030 ohms). The high-voltage right ventricular lead implanted in 2005 was a Medtronic 6944 Sprint Quattro ([Medtronic, Inc, Minneapolis, MN] R-wave 15.9 mV, pacing threshold 0.7 V at 0.5 milliseconds, and lead impedance 921 ohms).

After the procedure, the patient was comfortable. About 1 hour later, he went into respiratory distress with profuse sweating and pallor. His vitals were noted as pulse, 78/min regular; blood pressure, 130/88 mmHg; and respiratory rate, 18/min with pulse oximeter oxygen saturation at 98% on room air. A chest x-ray (CXR) showed a right apical pneumothorax about 3 cm in size (Figure 1). The patient’s distress continued, with falling oxygen saturation and blood pressure levels. His vitals deteriorated to pulse, 100/min regular; blood pressure, 100/60 mmHg; respiratory rate, 25/min; and pulse oximeter oxygen saturation, 92% on 5 L oxygen. A clinical evaluation suggested bilateral pneumothoraces. A repeat CXR revealed bilateral large pneumothoraces (Figure 2). A tension pneumothorax was imminent, and we opted for immediate insertion of bilateral chest tubes. The patient immediately improved; however, it should be noted that in the setting of suspected tension pneumothorax, a needle decompression typically is performed before a CXR. A repeat CXR showed full expansion of the patient’s lungs (Figure 3). He was discharged after 3 days. No subsequent pneumothorax was noted before the discharge and upon 2-week follow-up. Throughout the course of hospital stay during the procedure and postprocedure, the device was interrogated multiple times, and there was no evidence of lead malposition or atrium perforation.

DISCUSSION

This case is unique because it is the first of its kind to involve large bilateral pneumothoraces and no traumatic cardiac or lung injury. There was no direct evidence of atrial perforation or lead misplacement. So why did this patient develop bilateral large pneumothoraces?

In 2015, Rali and Manyam described a likely case of possible buffalo chest syndrome after ICD placement. This patient developed contralateral pneumothoraces necessitating surgical intervention four days after ICD placement. The authors suspected pleuropleural communication because the contralateral pneumothorax developed after ipsilateral chest tube clamping. The authors referred to a “buffalo chest” as a single pleural space with no anatomical separation of the two hemithoraces, as seen in an American buffalo or bison. In humans, the pleural cavities are separated, but a pleuropleural communication may result from intrathoracic procedures that lead to “iatrogenic buffalo chest.” Two similar cases had been reported. Both patients in these reports were noted to have contralateral pneumothoraces following an atrial rupture and misplaced atrial leads. In both of these cases, subclavian access could be obtained only after multiple attempts. In our patient, subclavian access was obtained without difficulty. There was a ventricular lead in place, and an atrial lead accompanied the ventricular lead. The atrial lead postprocedure remained in place with no changes in threshold and impedance. There was no direct evidence of atrial perforation. There also was no evidence of pericardial effusion, ruling out hemopericardium.

Several potential causes may have played a part in our case. The left-sided pneumothorax can be explained by a possible pleural rupture during subclavian access or atrial lead placement. Although we obtained subclavian access without difficulty, a pleural rupture can never be ruled out. However, the contralateral pneumothorax cause remained a mystery; pleuropleural communication may have been the trigger as previously reported. Another possible etiology is a right atrial microperforation. A common finding in all the reported cases (including ours) was use of an active fixation atrial lead. Although we did not see any obvious
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Difficult to Overrate

It would be difficult to overrate the value, as guides to practice, of the signs which declare themselves through the medium of the lungs in every case of unsound heart.

— Peter Mere Latham, MD, 1789-1875, British physician and medical educator, physician extraordinary to Queen Victoria

References