

## CASE RECORDS of the MASSACHUSETTS GENERAL HOSPITAL

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## Case 19-2015: A 71-Year-Old Man with Chest Pain and Shortness of Breath

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### PRESENTATION OF CASE

From the Departments of Medicine (D.M.D., L.M.P.), Radiology (A.M.P.), and Surgery (G.J.V.), Massachusetts General Hospital, and the Departments of Medicine (D.M.D., L.M.P.), Radiology (A.M.P.), and Surgery (G.J.V.), Harvard Medical School — both in Boston.

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*Dr. Katharine R. Clapham (Medicine):* A 71-year-old man with congestive heart failure and ischemic cardiomyopathy was admitted to this hospital because of sudden chest pain, diaphoresis, and shortness of breath.

The patient had hypertension, hyperlipidemia, diabetes mellitus, and ischemic cardiomyopathy and had a history of myocardial infarctions (an inferoposterior myocardial infarction, which had occurred 29 years earlier, and an apical infarction, which had occurred 14 years earlier and for which percutaneous balloon angioplasty of the first marginal branch of the left circumflex artery had been performed). Nine years before the current admission, a cardiac stress test with radionuclide imaging revealed a left ventricular ejection fraction of 33% and a reversible anterolateral defect; subsequent cardiac catheterization revealed severe disease in the right coronary artery and in branches of the left circumflex artery. Seven months before the current admission, echocardiography revealed that the left ventricular ejection fraction had worsened to 22%, and a subsequent cardiac stress test with radionuclide imaging (performed at this hospital) revealed a markedly dilated left ventricle, with large, dense inferior and lateral scars and associated minimal inferoapical ischemia. Eleven weeks before the current admission, transthoracic echocardiography (performed at this hospital) revealed a left ventricular ejection fraction of 23%, dilatation of the left atrium (anteroposterior dimension, 39 mm) and left ventricle (end diastolic dimension, 55 mm), diffuse hypokinesis of both ventricles (mild in the right and severe in the left), mild mitral and tricuspid regurgitation, and no pericardial effusion.

Three weeks later (8 weeks before the current admission), the patient was seen in the cardiac-arrhythmia clinic of this hospital to be evaluated for the placement of an implantable cardioverter–defibrillator (ICD). He reported exertional dyspnea that was consistent with a New York Heart Association (NYHA) functional class of II. He had no palpitations, presyncopal symptoms, syncopal events, orthopnea, or paroxysmal nocturnal dyspnea. His weight was stable. His medications were aspirin, irbesartan, carvedilol, hydrochlorothiazide, isosorbide mononitrate, atorvastatin, metformin, sitagliptin, and loratadine. He was allergic to penicillin and cephalosporins, which caused a rash, and he reported cough with the use of angio-

tensin-converting-enzyme inhibitors. He was married and had adult children. He had a smoking history of 45 pack-years but had stopped smoking 29 years earlier, and he had recently decreased alcohol consumption from 10 drinks per week to 4 on the advice of his clinicians; he did not use illicit drugs. Several relatives had diabetes mellitus.

On examination at that visit, the blood pressure was 110/70 mm Hg, the pulse 60 beats per minute, the respiratory rate 12 breaths per minute, and the body-mass index (the weight in kilograms divided by the square of the height in meters) 28.1. The jugular venous pressure was 6 cm of water; the remainder of the examination was normal, with no cardiac murmur or rub. Placement of an ICD was advised. Ten days before the current admission, a dual-chamber ICD system was inserted through the left axillary vein. Intravenous vancomycin was administered immediately before the procedure.

Immediately after the procedure, the patient was admitted to this hospital for routine monitoring. Chest radiographs that were obtained after the procedure and the following morning showed the ICD pulse generator on the left side of the chest, with ICD lead tips projecting over the right atrium and right ventricle, as well as clear and inflated lungs, mild enlargement of the cardiac silhouette (which was stable, as compared with the silhouette seen on earlier radiographs), and no pleural effusion or pneumothorax. Laboratory test results are shown in Table 1. The morning after ICD placement, repeat device interrogation was performed, and all measurements were within acceptable limits. The patient was discharged home later that day. During the next 9 days, he noted occasional mild positional pain at the ICD placement site that was relieved with acetaminophen. No skin changes or swelling at the ICD placement site were noted.

At 6 p.m. on the day of the current admission, midsternal chest discomfort and associated diaphoresis and mild dyspnea developed suddenly while the patient was watching television. The pain was exacerbated by movement and deep inspiration, did not radiate, was possibly improved by leaning forward, and was distinct from the pain at the ICD placement site that had been previously reported. The patient had no associated headache, light-headedness, palpitations, cough, nausea, vomiting, fever, or chills. He took two acetaminophen tablets for the pain, and after it

lasted for more than 1 hour, he sought medical attention. On arrival in the emergency department, approximately 3.5 hours after the onset of symptoms, the patient rated the pain at rest at 0 on a scale of 0 to 10, with 10 indicating the most severe pain. The midsternal pain could be reproduced with deep inspiration; he rated that pain at 7 of 10.

On examination, the temperature was 36.1°C, the blood pressure 130/70 mm Hg, the pulse 78 beats per minute and irregular, the respiratory rate 20 breaths per minute, and the oxygen saturation 96% while the patient was breathing ambient air. The skin was cool, with no inflammation or hematoma over the ICD placement site. Adhesive strips over the incision were intact. The first and second heart sounds were normal, with no murmur, and the breath sounds were slightly diminished at the bases. The jugular venous pressure was not recorded, and the remainder of the examination was normal. Cardiac telemetry showed sinus rhythm with occasional premature ventricular contractions. Blood levels of electrolytes, calcium, and phosphorus were normal; other test results are shown in Table 1.

*Dr. Anand M. Prabhakar:* Posteroanterior and lateral chest radiographs (Fig. 1) showed a new left pleural effusion, with no other clinically significant changes, as compared with the chest radiograph that was obtained immediately after the procedure. The cardiomeastinal silhouette was within normal limits, and the ICD leads appeared to be in a position similar to that on the previous radiograph.

*Dr. Clapham:* Cardiac ultrasonography, performed at the bedside by emergency department personnel, revealed trace pericardial effusion and decreased ventricular contractile function. An electrocardiogram (ECG) showed sinus rhythm at a rate of 66 beats per minute, with new ST-segment elevations in ECG leads II, III, aVF, V<sub>3</sub>, and V<sub>4</sub> (Fig. 2). Aspirin, magnesium sulfate, and insulin were administered. Twenty-six minutes later, approximately 3 hours after the patient's presentation, severe pain recurred. Fentanyl was administered intravenously. Approximately 30 minutes after the onset of this pain, light-headedness, dizziness, diaphoresis, nausea, and transient hypotension (blood pressure, 67/44 mm Hg) occurred, with a pulse of 59 beats per minute. These symptoms spontaneously improved within minutes; the systolic blood pressure rose to 92 mm Hg and subsequently ranged between 80 and 92 mm Hg,

**Table 1. Laboratory Data.\***

| Variable  | Reference Range, Adults† | On First Admission,<br>1 Day after<br>ICD Implantation | On Current Admission,<br>10 Days after<br>ICD Implantation |
|---|--------------------------|--|--|
| Hematocrit (%)  | 41.0–53.0                | 42.6   | 39.0   |
| Hemoglobin (g/dl)   | 13.5–17.5                | 14.6   | 13.5   |
| White-cell count (per mm <sup>3</sup> )                               | 4500–11,000              | 5900   | 12,000   |
| Differential count (%)  |                          |  |  |
| Neutrophils   | 40–70                    | 69.5   | 73.8   |
| Lymphocytes   | 22–44                    | 17.0   | 13.5   |
| Monocytes   | 4–11                     | 8.5  | 8.4  |
| Eosinophils   | 0–8                      | 4.6  | 3.6  |
| Basophils   | 0–3                      | 0.2  | 0.2  |
| Platelet count (per mm <sup>3</sup> )                                 | 150,000–400,000          | 134,000  | 192,000  |
| Prothrombin time (sec)  | 11.0–14.0                |  | 14.2   |
| Prothrombin-time international<br>normalized ratio                    | 0.9–1.1                  |  | 1.1  |
| Urea nitrogen (mg/dl)   | 8–25                     | 18   | 23   |
| Creatinine (mg/dl)  | 0.60–1.50                | 0.97   | 1.36   |
| Estimated glomerular filtration rate<br>(ml/min/1.73 m <sup>2</sup> ) | ≥60                      | >60  | 52   |
| Glucose (mg/dl)   | 70–110                   | 139  | 290  |
| Magnesium (mg/dl)   | 1.7–2.4                  | 1.7  | 1.6  |
| Troponin T (ng/ml)  | <0.03                    |  | <0.01  |

\* ICD denotes implantable cardioverter–defibrillator. To convert the values for urea nitrogen to millimoles per liter, multiply by 0.357. To convert the values for creatinine to micromoles per liter, multiply by 88.4. To convert the values for glucose to millimoles per liter, multiply by 0.05551. To convert the values for magnesium to millimoles per liter, multiply by 0.4114.

† Reference values are affected by many variables, including the patient population and the laboratory methods used. The ranges used at Massachusetts General Hospital are for adults who are not pregnant and do not have medical conditions that could affect the results. They may therefore not be appropriate for all patients.

and the pulse ranged between 57 and 64 beats per minute. Vancomycin was administered. Five hours after presentation, the patient was admitted to the cardiology service. On arrival, the blood pressure was 120/55 mm Hg, and the pulse was 65 beats per minute, with an inspiratory decline in systolic pressure of less than 5 mm Hg. Breath sounds were diminished at the left base, the jugular venous pressure was 9 cm of water, and there were normal heart sounds, with no rubs or murmurs; the remainder of the examination was unchanged. A repeat chest radiograph was unchanged, as compared with a radiograph obtained on admission.

Approximately 13 hours after the patient's presentation, diagnostic procedures were performed.

#### DIFFERENTIAL DIAGNOSIS

*Dr. David M. Dudzinski:* Ten days after this 71-year-old man had undergone ICD placement, he presented with acute pleuritic chest pain, intermittent hypotension, new inferior and anterior ST-segment elevations on ECG, clinical and radiographic findings suggesting a new left pleural effusion, and evidence of a new pericardial effusion on bedside ultrasonography. The patient also had intermittent hypotension, with the

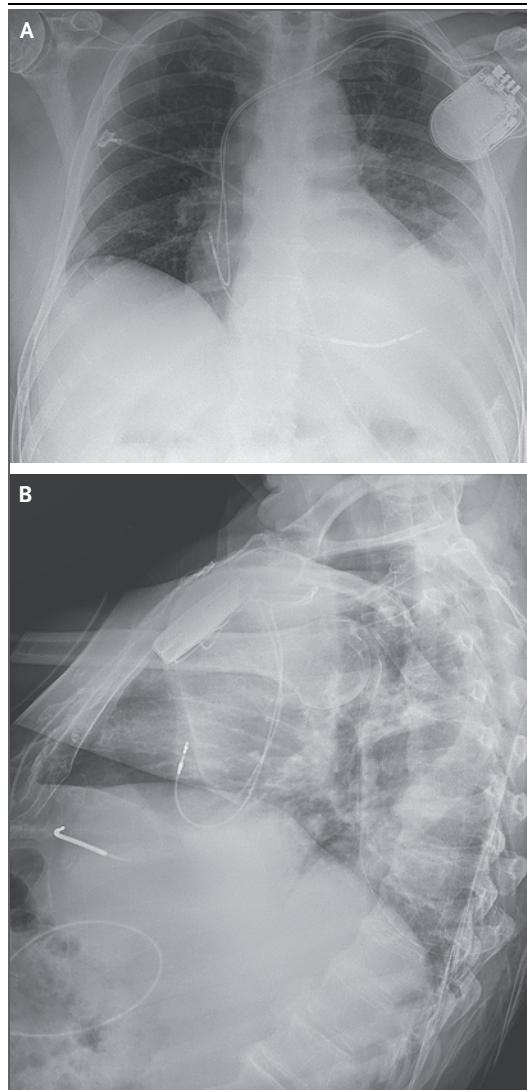
overall syndrome developing rapidly. A number of possibilities can explain this constellation of findings; I will consider these and highlight likely and potentially lethal diagnoses.

#### MYOCARDIAL INFARCTION

Given this patient's history of ischemic heart disease and multiple cardiac risk factors, the sudden onset of chest pain and new ST-segment elevations raise concerns that he may have had an acute myocardial infarction. Hypotension can result from an anterior or inferior myocardial infarction due to incremental left ventricular systolic dysfunction, a concomitant right ventricular infarction in a patient with an inferior myocardial infarction, or a mechanical complication of a subacute infarction, such as papillary muscle rupture. A right ventricular infarction is unlikely in this case because no clinically significant ST-segment elevations were present in ECG leads  $V_1$  or  $V_{4R}$  and because Kussmaul's sign (the paradoxical rise in jugular venous pressure with inspiration)<sup>1</sup> was not reported. The presence of new inferior and anterior ST-segment elevations, spanning multiple coronary territories, may itself argue against an acute ischemic event. It is important to note that, although the patient had a negative test for troponin T on presentation (3.5 hours after the onset of symptoms), this result does not necessarily rule out myocardial infarction. The pleuritic nature of this patient's chest pain makes an acute coronary syndrome unlikely.<sup>2</sup> This patient also presented with maximal intensity of pain at onset; patients with ischemic pain classically present with a crescendo temporal pattern of pain, whereas patients with aortic, pericarditic, or pleuritic pain are more likely to present with maximal intensity at onset.<sup>2</sup>

#### ACUTE AORTIC SYNDROME

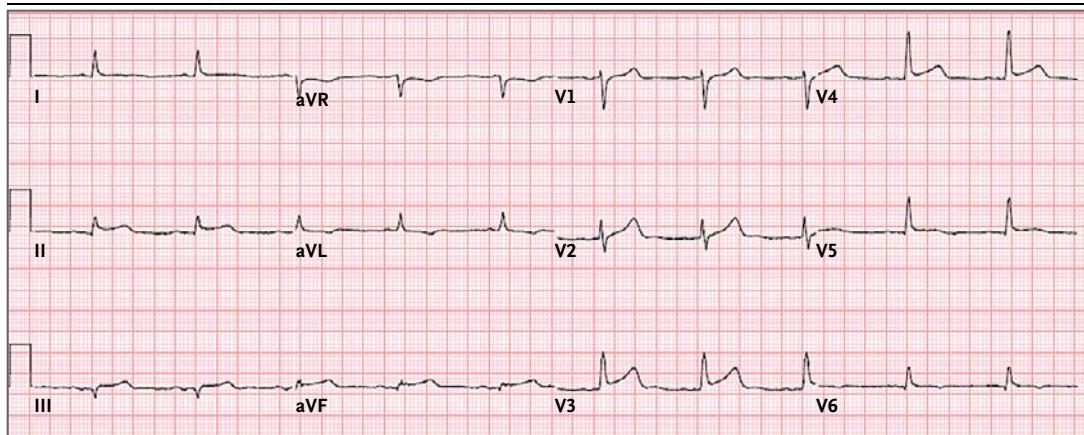
An acute aortic syndrome of the ascending aorta can result in a dissection in the right coronary artery; this could produce the inferior ST-segment elevations seen on ECG in this case and a hemorrhagic pericardial effusion, which could have caused the patient's hypotension and pleuritic pain. It is unclear why this patient would have pleuritic pain due to a complication of dissection but not the severe or tearing pain in the chest or back that is characteristic of aortic dis-



**Figure 1. Chest Radiographs.**

Posteroanterior and lateral chest radiographs (Panels A and B, respectively), obtained on the current admission, show a new left pleural effusion.

section. Nevertheless, the patient's pulse pressure may have widened (from 110/70 mm Hg to 120/55 mm Hg), raising the possibility that he had acute aortic insufficiency, which may be seen in patients with dissection.<sup>3</sup> The equal blood pressure in both arms, symmetric radial pulses, absence of murmur associated with aortic insufficiency, and sharp and nonwidened mediastinum on radiography all weigh against a diagnosis of acute aortic syndrome in this patient. Perhaps



**Figure 2. Electrocardiogram.**

An electrocardiogram, obtained on the current admission, shows sinus rhythm with 1-mm, concave ST-segment elevations in the inferior leads (II, III, and aVF) and in leads V<sub>3</sub> and V<sub>4</sub>, along with T-wave inversions and flattening in the lateral leads. The changes in ST-segment elevations are new, as compared with the tracing obtained 10 days earlier.

the most compelling factor that argues against this diagnosis is the absence of pain at rest 3.5 hours after symptom onset.

#### PLEURITIS AND PERICARDITIS

Other cardiopulmonary conditions that may cause pleuritic chest pain include pneumothorax, pneumonia, pleuropericarditis, and pulmonary embolism. Pneumothorax is a known complication of percutaneous implantation of electrophysiological devices, owing to the proximity of the access veins to the lung apices. In this patient, the ICD was placed through the axillary vein, and pneumothorax more commonly occurs when the device is placed through the subclavian vein.<sup>4</sup> In addition, pneumothorax was not detected on either of the two radiographs that were obtained immediately after device placement, and it would be unusual for a hemodynamically significant procedure-related pneumothorax to first manifest 10 days after placement. A spontaneous pneumothorax is possible in this patient given his long smoking history, although primary spontaneous pneumothorax usually occurs in the second to fourth decades of life, and this patient is 71 years of age<sup>5</sup>; furthermore, he has no known history of pulmonary disease that would increase the likelihood of pneumothorax. Pneumothorax may produce such ECG findings as ST-segment elevations, and tension pneumothorax may impair venous return and thereby cause hypoten-

sion. A pleural effusion can occur concurrently with pneumothorax, but it is not clear how a pericardial effusion would have developed in this patient.

Pneumonia with a parapneumonic effusion is another possible cause of this patient's illness, given the mild leukocytosis and pleuritic pain. Although bacteremia and sepsis may cause hypotension, infection alone does not account for the changes on ECG and ultrasonography that were seen in this case.

Pulmonary embolism may cause acute pleuritic pain, hypotension, and pleural effusion, and affected patients may occasionally present with inferior or precordial ST-segment elevations on ECG.<sup>6</sup> Pulmonary embolism may result from leg-vein thrombosis after a recent procedure and brief hospitalization or from arm-vein trauma and local stasis after introduction of transvenous wires during ICD placement. The absence of hypoxemia, tachycardia, and right ventricular strain make a pulmonary embolism unlikely in this case.

Does this patient have pleuropericarditis? Acute inflammation of both the pericardial and pleural surfaces may cause pleuritic pain and diffuse ST-segment elevation on ECG. Serosal inflammation often results in accumulation of transudative fluid, which can cause pericardial and pleural effusions. The ST-segment elevation associated with acute pericarditis is typically accompanied by PR-segment depression, which was not observed

in this case.<sup>7</sup> It is unclear whether a new primary serositis would produce enough pleural fluid to be detectable on examination. In addition, most causes of diffuse serositis are associated with an indolent presentation, which is not consistent with this patient's acute presentation.

#### COMPLICATIONS OF ICD IMPLANTATION

Could the features of this patient's presentation be attributed to the ICD placement? Complications of ICD placement are well described, and ICD lead migration or dislodgment occurs within a few days after implantation in approximately 0.14 to 1.2% of patients<sup>8-11</sup>; the overall incidence of ICD lead dislodgment is highest in the first weeks after implantation, before myocardial fibrosis occurs at the site of the tip of the ICD lead. In nearly 11% of patients with lead dislodgment, another related major adverse event (e.g., cardiac perforation and tamponade, pneumothorax, or cardiac arrest) or in-hospital death occurs.<sup>9</sup> It is important to note that chest radiography is not very sensitive in the detection of lead migration. However, when we compare the chest radiographs obtained on the current admission with radiographs obtained 1 day after ICD implantation (not shown), there does appear to be a subtle difference in the positions of the leads with respect to the cardiac silhouette. Right ventricular myocardial perforation would generate inferior ST-segment elevations as a manifestation of direct cardiac injury. The immediate physiological result of free-wall perforation is hemopericardium, which would account for the finding of pericardial effusion on ultrasonography and the pleuritic pain. Hypotension could be the result of cardiac tamponade due to pericardial effusion, but this is unlikely in a patient who has a relatively normal jugular venous pressure, no pulsus paradoxus, and relative bradycardia (although this patient was receiving carvedilol, which may blunt compensatory tachycardia). Of note, tamponade physiology may be associated with large pleural effusions.<sup>12</sup> Depending on the anatomical trajectory of the dislodged lead, intrathoracic structures such as the pleura and lungs may be perforated, resulting in hemothorax with hypotension related to brisk blood loss; however, an increasing jugular venous pressure argues against a diagnosis of volume depletion.

During the diagnostic evaluation of a patient who has had any recent medical or surgical pro-

cedure, the clinician should consider and rule out periprocedural complications. The manifestations of ICD lead migration are protean and may be surprisingly subtle; in fact, both the overall examination and initial bedside ultrasound examination in this case may be deceptively interpreted as close to normal. However, the events after a lead perforation may evolve rapidly, and a normal overall examination or ultrasound examination at any one point in time cannot rule out a perforation. To make a diagnosis of lead perforation, a high index of suspicion is required, and the diagnostic strategy must account for all these factors and expeditiously rule out other lethal possibilities, including aortic dissection and pulmonary embolism. I believe that the most likely diagnosis in this case is ICD lead migration and subsequent perforation of the myocardium and pleura, resulting in hemopericardium and hemothorax. This diagnosis neatly ties together all the signs and symptoms associated with this patient's presentation. In order to confirm the diagnosis, I would perform axial computed tomography (CT) to define the lead position, repeat device interrogation to confirm changes in lead impedance and capture threshold, and formal echocardiography to assess for pericardial effusion and evidence of tamponade physiology.<sup>13,14</sup>

*Dr. Eric S. Rosenberg (Pathology):* Dr. Ptaszek, what was your impression when you evaluated this patient?

*Dr. Leon M. Ptaszek:* Although there were numerous possible explanations for this patient's chest pain, we were most concerned about the possibility of a procedural complication. In particular, we were worried that the ICD lead had migrated from its implantation site and perforated the myocardium, resulting in pericardial and pleural effusions. Lead perforation must be addressed promptly, because it can precipitate life-threatening cardiac tamponade within minutes. Early clinical signs of lead perforation can be subtle and nonspecific, so a rapid and focused evaluation is required, even in the absence of signs of tamponade on physical examination.

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#### CLINICAL DIAGNOSIS

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Myocardial perforation by an implantable cardioverter-defibrillator lead, causing pericardial and pleural effusions.

**Table 2.** Measurements for Implantable Cardioverter–Defibrillator Leads.

| Variable                 | Normal                      | On First Admission,<br>1 Day after<br>ICD Implantation | On Current Admission,<br>10 Days after<br>ICD Implantation     |
|--------------------------|-----------------------------|--|--|
| Cardiac electrogram (mV) | >4                          | 11.2   | 1.5  |
| Lead impedance (ohms)    | 300–1200                    | 640  | 240  |
| Capture threshold        | Capture<br>(output, <1.5 V) | Capture<br>(output, 0.5 V;<br>pulse width, 0.4 msec)   | Failure to capture<br>(output, 10 V;<br>pulse width, 1.5 msec) |

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DR. DAVID M. DUDZINSKI'S  
DIAGNOSIS

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Migration of an implantable cardioverter–defibrillator lead, resulting in myocardial perforation, hemopericardium, and hemothorax.

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IMAGING AND  
ELECTROPHYSIOLOGICAL STUDIES

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*Dr. Rosenberg:* Dr. Ptaszek, what did your evaluation show?

*Dr. Ptaszek:* Routine device interrogation that was performed the morning after ICD implantation revealed that all lead measurements were within normal limits. Repeat device interrogation on the current admission revealed several abnormal measurements (Table 2). The voltage of the cardiac electrogram that was sensed by the electrode at the tip of the ventricular lead had fallen by nearly an order of magnitude since implantation. In addition, the lead impedance had decreased considerably since implantation. Pacing of the ventricle could not be achieved, even with maximum output. Together, these findings are consistent with compromised contact between the lead tip and the myocardium. The next step in the evaluation of this patient was to obtain a CT scan of the chest to determine whether the tip of the ventricular lead had migrated through the ventricular wall. Dr. Prabhakar, would you show us the CT scans?

*Dr. Prabhakar:* Selected axial and reformatted CT images of the chest, obtained without the administration of contrast material, showed a high-density left pleural effusion, with an ICD lead perforating the pericardium and the adjacent fat. The high-density left pleural effusion is consistent with a hemothorax (Fig. 3).

*Dr. Ptaszek:* Although the presence of the ventricular lead tip outside the myocardial wall was confirmed by the CT scan, lead migration cannot be managed on the basis of imaging findings alone. Careful correlation between the imaging findings and repeat device interrogation is required before a treatment strategy can be formed. It is possible to see a lead tip located beyond the myocardial border on CT without finding any evidence of change in lead measurements or pericardial effusion; this is frequently termed an asymptomatic lead perforation and does not necessarily require revision of the lead.<sup>15</sup> The changes in lead measurements that were seen in this case can also be observed if the lead detaches from the myocardium but remains inside the heart. It is also important to remember that changes in lead measurements can reveal lead migration even in the absence of definitive imaging findings.

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DISCUSSION OF MANAGEMENT

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*Dr. Gus J. Vlahakes:* The key issue in the management of a lead perforation is to be prepared for decompensation or a disaster at the time the lead is extracted. Extraction of a migrated lead is performed in the operating room; the patient should receive general anesthesia and be monitored with transesophageal echocardiography. In the majority of cases, a lead associated with a perforation can be withdrawn without substantial bleeding into the pericardium. In this case, since the patient had an ICD lead with a large diameter and blood in the pericardium that needed to be evacuated, I prepared and draped the patient in case the sternum needed to be opened. I opened the subxiphoid part of the chest; with retraction of the diaphragm toward the feet and retraction of the sternum anteriorly, the ICD lead

could be easily seen perforating the apex of the right ventricle and an overlying band of epicardial fat. The lead tip, which had penetrated the pericardium and epicardial fat, had also burrowed into the pleural space and allowed approximately 1 unit of blood to collect in the left side of the chest. The migrated lead was placed on traction and cut; we had planned to have the electrophysiology team remove the old lead and replace it with a new one the next day. When the ICD lead was pulled back into the heart, the myocardial exit site spontaneously sealed. To ensure that it was completely sealed, I reinforced the exit site with a suture. Blood was then evacuated from both the pericardium and the left pleural space, and tube drainage was established at each site. The surgical incision was closed. The patient tolerated the procedure well, with no adverse events.

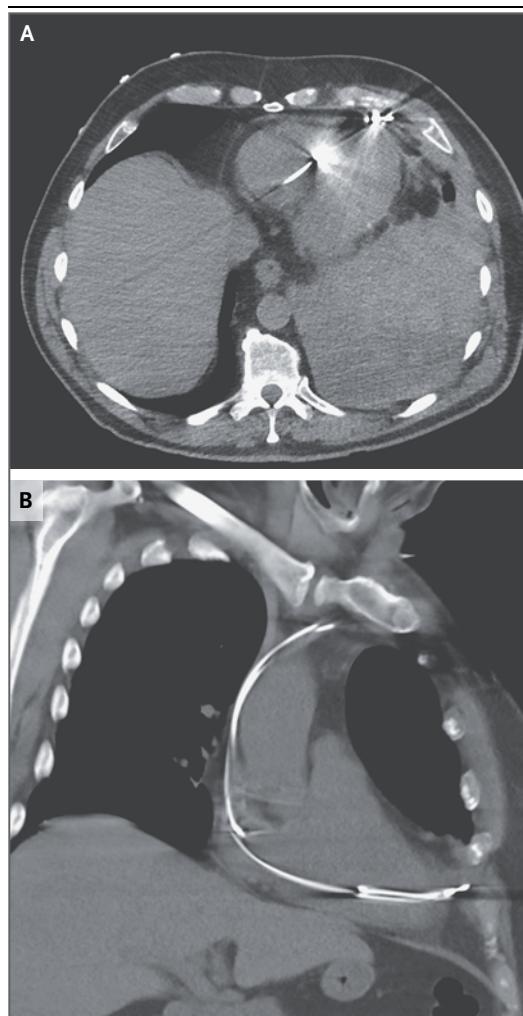
*Dr. Clapham:* After the surgical procedure, the patient was transferred to the cardiac surgical intensive care unit and, shortly thereafter, to the step-down unit. On postoperative day 2, he underwent revision of the ICD system, with transvenous removal of the old right ventricular ICD lead and placement of a new lead; he went home on postoperative day 4. At a clinic visit 1 month after discharge, the patient reported feeling mildly fatigued but otherwise well. He walks 1 to 2 miles several days a week.

*Dr. Hasan Bazari (Medicine):* Is this patient's history of ischemic cardiomyopathy a contraindication for device implantation?

*Dr. Ptaszek:* No. According to extensive registry data, there is a clear benefit associated with ICD implantation in a patient who has ischemic cardiomyopathy with NYHA class II symptoms (despite maximal medical therapy) and a left ventricular ejection fraction of 35% or less. This risk–benefit relationship was determined on the basis of outcomes in patients not unlike the patient described in this case.

*Dr. Lloyd Axelrod (Medicine):* Are there known risk factors for lead perforation?

*Dr. Ptaszek:* A few risk factors for lead perforation, including female sex and a low body-mass index, have been described. Some data suggest that myocardial fibrosis, which is frequently observed in patients with ischemic cardiomyopathy, may be protective against perforation. Ven-



**Figure 3. CT Images of the Chest.**

Axial and reformatted CT images of the chest (Panels A and B, respectively), obtained without the administration of contrast material, show a left hemothorax and an ICD lead that is perforating the pericardium and the surrounding pericardial fat.

tricular hypertrophy and diabetes, both of which are associated with fibrosis, may be associated with reduced rates of perforation.<sup>15</sup>

*A Physician:* At what point do you stop worrying that a perforation could occur?

*Dr. Ptaszek:* Perforation is most likely to occur within days to weeks after device implantation. Perforations that occur 1 month or more after implantation are rare but have been reported.

## FINAL DIAGNOSIS

## Perforation of the right ventricular wall by an implantable cardioverter-defibrillator lead.

This case was presented at the Medical Case Conference. No potential conflict of interest relevant to this article was reported.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

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## LANTERN SLIDES UPDATED: COMPLETE POWERPOINT SLIDE SETS FROM THE CLINICOPATHOLOGICAL CONFERENCES

Any reader of the *Journal* who uses the Case Records of the Massachusetts General Hospital as a teaching exercise or reference material is now eligible to receive a complete set of PowerPoint slides, including digital images, with identifying legends, shown at the live Clinicopathological Conference (CPC) that is the basis of the Case Record. This slide set contains all of the images from the CPC, not only those published in the *Journal*. Radiographic, neurologic, and cardiac studies, gross specimens, and photomicrographs, as well as unpublished text slides, tables, and diagrams, are included. Every year 40 sets are produced, averaging 50-60 slides per set. Each set is supplied on a compact disc and is mailed to coincide with the publication of the Case Record.

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