

MINI-FOCUS ISSUE: ELECTROPHYSIOLOGY

INTERMEDIATE

CASE REPORT: CLINICAL CASE: PROCEDURAL COMPLICATIONS

Fatal Spontaneous Intercostal Artery Bleeding During Catheter Ablation for Atrial Fibrillation



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ABSTRACT

Serious adverse events such as hemothorax are rarely seen in catheter ablation for atrial fibrillation. A recent case report discussed hemothorax from injury of the intercostal artery during atrial fibrillation ablation. Our case presents a patient with spontaneous bleeding from the intercostal artery that led to hemothorax, disseminated intravascular coagulation, and death. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2022;4:101614)
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HISTORY OF PRESENTATION

A 59-year-old Asian woman presented to our electrophysiology lab for a repeat ablation due to continued symptomatic atrial fibrillation (AF) episodes despite being on dofetilide. In the morning of her presentation, her laboratory tests were unremarkable, including normal hemoglobin/hematocrit (13.4 g/dL/41.5%) and platelet (190 k/ μ L), and slightly prolonged prothrombin time (15.3 sec), partial thromboplastin time (42 seconds), and mildly elevated international normalized ratio (1.3) in the setting of being on apixaban. Presentation electro-

cardiogram showed sinus bradycardia with incomplete right bundle branch block and nonspecific T-wave changes. The patient underwent general anesthesia with etomidate, rocuronium, versed, fentanyl, and propofol. Femoral venous access was obtained under ultrasound guidance, and heparin was given to achieve the target activated clotting time >300 seconds. The dose was calculated based on body weight (100 units/kg bolus injection). After the first transseptal access, the patient was hypotensive but responded quickly to a push of phenylephrine and fluids. Right lung expansion was noted to be reduced after the second transseptal access. Intracardiac echo did not show any pericardial effusion. The first activated clotting time was within the range of 300 to 350 seconds. By this time, we asked our anesthesiologist to check the endotracheal tube position to ensure that both lungs were adequately ventilated. After approximately 10 minutes of ablation time in the left atrium, the patient was hypotensive requiring more pressor support. Right lung motion was nearly absent

LEARNING OBJECTIVES

- To recognize hemothorax during AF ablation procedure and proceed with appropriate workups.
- To understand the best management strategy for spontaneous ICA bleeding.

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the [Author Center](#).

Manuscript received July 18, 2022; revised manuscript received August 5, 2022, accepted August 10, 2022.

**ABBREVIATIONS
AND ACRONYMS**

AF = atrial fibrillation
CT = computed tomography
DIC = disseminated intravascular coagulation
ICA = intercostal artery

and increased pleural effusion was seen under fluoroscopy. At this point, heparin was reversed with protamine, and coagulation factors and blood products were ordered.

PAST MEDICAL HISTORY

The patient had a history of hypertension, obstructive sleep apnea, and paroxysmal AF with prior pulmonary vein isolation and cavotricuspid isthmus ablation.

DIFFERENTIAL DIAGNOSIS

In the setting of transeptal access and hemodynamic compromise, perforation of cardiac chambers causing tamponade is an obvious concern. Transeptal access resulting in hemothorax is extremely rare but not impossible.

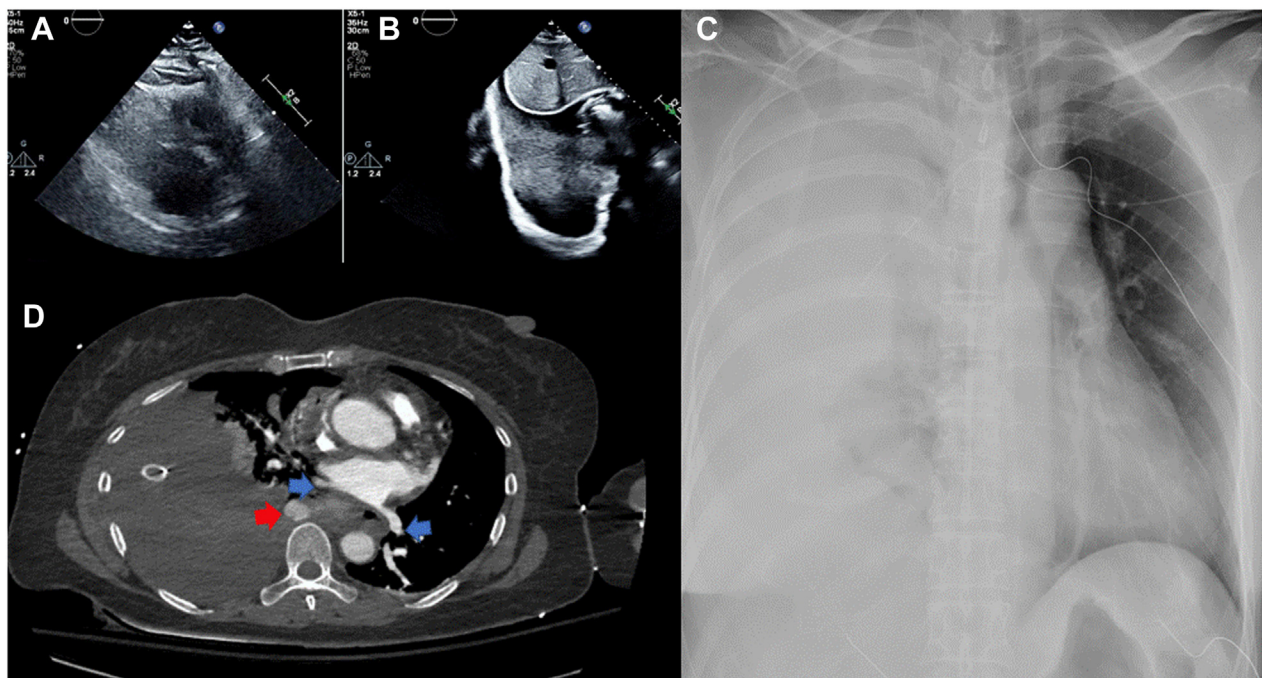
INVESTIGATIONS

Stat transthoracic echocardiogram showed trivial anterior pericardial effusion and large right hemothorax (Figures 1A and 1B). Stat chest x-ray confirmed

large right pleural effusion causing complete atelectasis of right lung and leftward mediastinal shift (Figure 1C). A chest tube was emergently placed and drained 2.3 L of blood. With massive transfusion protocol, the patient's hemodynamics stabilized, and she was eventually extubated in the intensive care unit. Laboratory tests showed hemoglobin/hematocrit of 7.9 g/dL/24.2%, platelet of 134 k/ μ L, arterial blood gas pH of 7.17, lactate of 6.4 mmol/L, and adequate oxygenation. Computed tomography (CT) angiogram of chest, abdomen, and pelvis noted evidence of brisk active bleeding along the posterior margin of the heart and the right lateral margin of the esophagus (Figure 1D).

MANAGEMENT

After returning from CT, she became hemodynamically unstable and was re-intubated. The cardiothoracic surgery team brought her to the operating room for exploration of the right pleural space. High-volume bleeding was noted from the inferior pleural cavity in the region of the right paraspinous area and eventually achieved adequate hemostasis. There

FIGURE 1 Hemothorax on Various Imaging Modalities

(A) Short-axis view of echocardiography showing trivial anterior pericardial effusion. (B) Subcostal view of echocardiography showing right hemothorax. (C) Chest x-ray shows large right pleural effusion that compresses the right lung and causes leftward mediastinal shift. (D) Axial cut of computed tomography with contrast at the level of the left atrium. Blue arrows point to left and right inferior pulmonary veins and the red arrow points to bleeding along the posterior margin of the heart.

was no evidence of injury in the left atrium and all 4 pulmonary veins. During the surgery, significant and continued bleeding was noted from all surfaces consistent with coagulopathy. Her intraoperative platelet count was 6,000, and she was given numerous doses of clotting factors, platelets, and blood. After initial closure of her chest, she continued to have moderate to excessive bloody output from the right pleural space and re-exploration showed bleeding from all surfaces. Quick clot packing material was placed and chest tub output seemed to improve. The patient was taken back to the intensive care unit and interventional radiology was called for embolization of a bleeding vessel. Unfortunately, despite continued resuscitation, she was maxed out on multiple pressors and coded several times. Her laboratory tests were consistent with disseminated intravascular coagulation (DIC). She coded in transit to the interventional radiology suite and died.

DISCUSSION

We present a case of intercostal artery (ICA) bleeding leading to hemothorax, DIC, and death in the setting of AF ablation. A recent case report discusses 2 similar cases in which it was thought to be from injury by the ablation itself.¹ The mechanism of ICA injury was theorized to be from heat energy or mechanical pressure of the catheter that reached the ICA, and the case report also discusses the anatomic location of 8 right ICAs as the most vulnerable site of injury of all ICAs. However, in our case, we noticed right pleural effusion even before any ablation was performed, which suggests that this entity may not be related to the ablation. Surgical exploration and autopsy data confirmed that there was no evidence of perforation of any cardiac chambers or pulmonary veins, and bleeding was from the right 8th ICA. Unlike the previously published case report, our patient did not survive due to DIC and hemorrhagic shock. The entity of spontaneous ICA bleeding has been previously reported to be associated with various medical conditions, such as neurofibromatosis type 1, systemic lupus erythematosus, coarctation of aorta, cirrhosis, spontaneous pseudoaneurysm, and spinal procedures including acupuncture.²⁻⁶ Our patient had no history

of these conditions, and it is presumed to be spontaneous.

Thoracotomy and, in certain cases, angiographic embolization can be an option for treatment. Although most literature on angiographic coil embolization is based on trauma-related ICA injury, several case reports suggest it is safe and effective in spontaneous ICA bleeding.⁷⁻⁹ The seemingly best management strategy for spontaneous rupture of the ICA appears to be a combination of coil embolization and surgical repair.^{1,8}

FOLLOW-UP

Unfortunately, DIC and severe hemorrhagic shock precluded our patient's survival. The autopsy result is as follows: There is no evidence of perforation of the chambers of the heart or great vessels including the pulmonary veins. There is no intraparenchymal hematoma of the lung. Bleeding is from the right eighth ICA.

CONCLUSIONS

Even though right hemothorax was recognized early in the procedure with guidance of fluoroscopy and echocardiography, ICA bleeding presents a clinical challenge due to high mortality.¹⁰ CT with contrast was not particularly helpful in identifying the exact source of bleeding in our case. Because previous case report patients and our patient are East Asians, we wonder if patients with East Asian ethnicity are at higher risk of developing spontaneous ICA bleeding. We will need further case series to identify the exact mechanism and risk factors of this entity as well as its relation to AF ablation.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS atrial fibrillation, catheter ablation, intercostal artery bleeding