

Severe hemothorax complications during atrial fibrillation ablation: Lessons from two cases



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Introduction

Atrial fibrillation (AF) ablation has rapidly become a safe and effective treatment method owing to recent advancements in technology and more experienced operators.¹ However, there is a risk of rare but severe complications of AF ablation,^{2,3} such as cerebral infarction, cardiac tamponade, pulmonary vein (PV) stenosis, or atrioesophageal fistula. One of such severe complications, hemothorax, is rare. A previous worldwide survey reported an incidence of 0.02%.¹ This study reports 2 cases of hemothorax due to AF ablation, both associated with bleeding of the intercostal artery (ICA); presents the clinical course of this complication; and discusses the treatment and prevention.

Case report

All procedures performed in this study were in accordance with the ethical standards of the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards, and the study was approved by the Institutional Research Committee of Tokyo Heart Rhythm Clinic. Written informed consent was obtained from the patients for the publication of this study.

Case 1

A 63-year-old man was admitted to our hospital for congestive heart failure due to AF-induced cardiomyopathy. The patient returned to sinus rhythm with oral amiodarone (100 mg per day). At 1 month after improvement in heart failure, we performed PV isolation by 3-dimensional (3D) mapping-guided radiofrequency catheter ablation under conscious sedation. Bilateral PV isolation was achieved after completion of the index-wide antral circumferential ablation around

KEY TEACHING POINTS

- Right hemothorax during atrial fibrillation ablation was caused by bleeding from the right eighth intercostal artery.
- There is a risk of intercostal artery injury during isolation of the pulmonary vein, especially of the inferior part to the posterior wall side of the right inferior pulmonary vein.
- Contrast-enhanced computed tomography can precisely identify the location of the intercostal artery and thus be useful to assess the risk of this complication.
- For early detection of pleural effusion, fluoroscopy and echocardiography should be performed during the procedure.

the PV. However, the right inferior PV required an additional ablation on the posterior wall of the carina owing to the acute PV reconnection (Figure 1A). The contact force, radiofrequency power, and its delivery time were 5–20 g, 25 W, and 20–30 seconds, respectively, during PV isolation. In particular, the point-by-point lesion parameters of the ablation for the inferior part to the posterior wall side of the right inferior PV were as follows: average contact force, 11.5 ± 2.6 g; radiofrequency power, 25 W; and delivery time, 25 ± 2 seconds. Immediately after the procedure, the patient complained of pain in the right back. Contrast-enhanced computed tomography (CT) revealed right hemothorax and contrast leakage from the right ICA (Figure 1B). We diagnosed hemothorax caused by bleeding from the right eighth ICA and performed emergency coil embolization (Figure 1C). The patient complained of dyspnea the next day, and contrast-enhanced CT revealed that the size of the hematoma had increased in the right chest wall, suggesting rebleeding (Figure 1D). We attempted surgical hemostasis assisted by thoracoscopy but found no active bleeding after removing the hematoma from the right chest wall. Thereafter,

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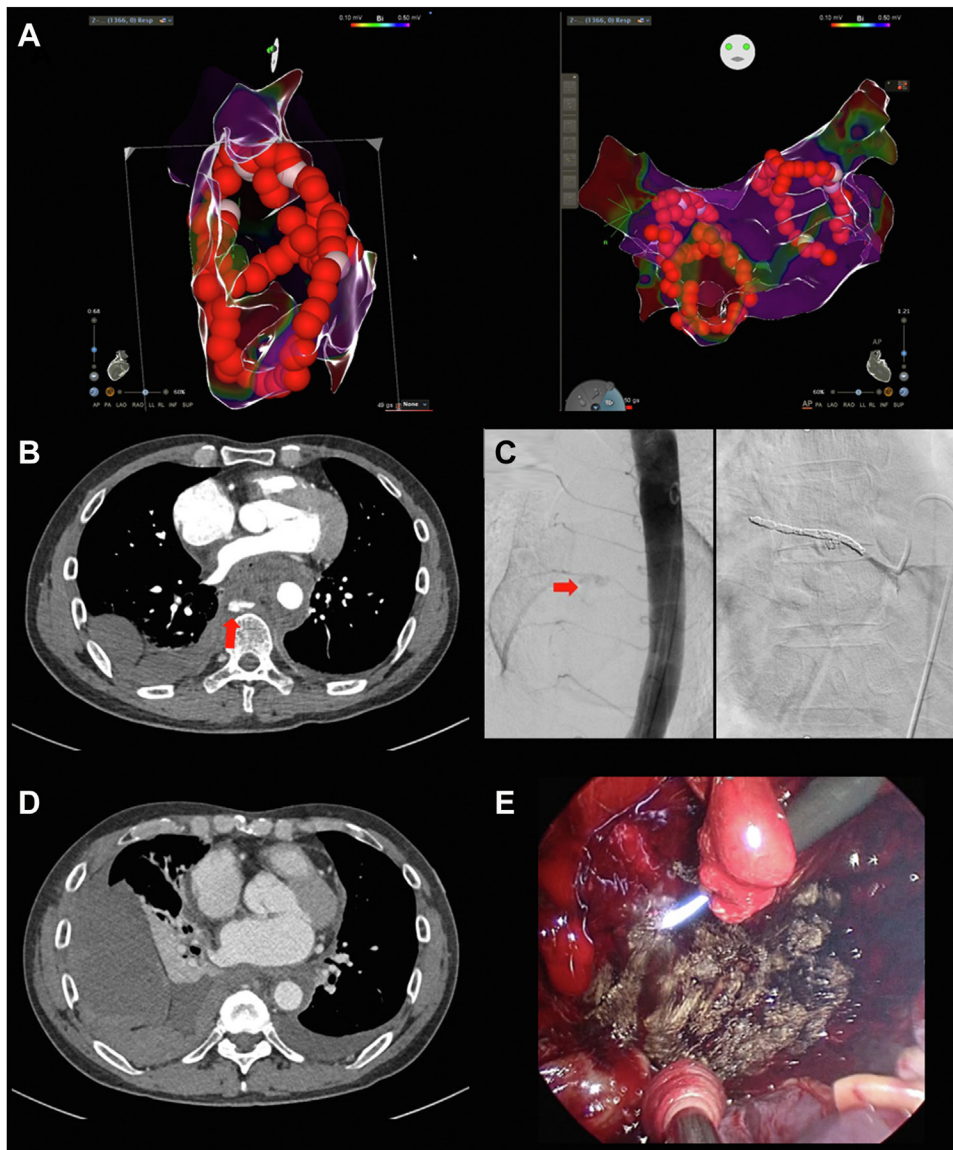


Figure 1 **A:** Voltage map with lesion tags during pulmonary vein isolation in case 1: inner view of right pulmonary vein and front view of left atrium. **B:** Contrast-enhanced computed tomography of the hematoma and contrast leakage from the right intercostal artery (*red arrow*). **C:** Aortography demonstrating extravasation from the right eighth intercostal artery (*red arrow*) and digital subtraction image after embolization of the right eighth intercostal artery with coils. **D:** Contrast-enhanced computed tomography of an enlarged hematoma within the right chest wall. **E:** Thoracoscopic removal of intrathoracic hematoma and coagulation therapy with argon plasma.

we induced coagulation therapy with argon plasma to stop the oozing from the chest wall (Figure 1E). After surgical hemostasis, the patient reported no rebleeding, the hemodynamics remained stable, and the hemoglobin level was retained at 9–11 g/dL, with red blood cell transfusion of 1120 mL. Heparin was initiated on day 11 to keep the activated clotting time at 150–200 seconds. The patient resumed edoxaban tosylate hydrate (30 mg) on day 18, and we verified no hemorrhagic complications during the 4 days after resumption of oral anticoagulant. The patient was discharged from the hospital on day 22 without sequelae. The patient has experienced no chest symptoms or recurrence of AF during the 6-month follow-up period.

Case 2

A 55-year-old man with a history of cerebral infarction was referred for curative treatment of paroxysmal AF. The AF burden was 46% on 24-hour ambulatory monitoring performed before admission. Other laboratory findings, including chest radiography, echocardiography, and blood tests, were all unremarkable. We performed PV and superior vena cava isolation by 3D mapping–guided radiofrequency catheter ablation under general anesthesia. We isolated both sides of the PV after completion of the index-wide antral circumferential ablation around the PV (Figure 2A). During PV isolation, the contact force, radiofrequency power, and its delivery time were 5–20 g, 30–40 W, and 15–40 seconds,

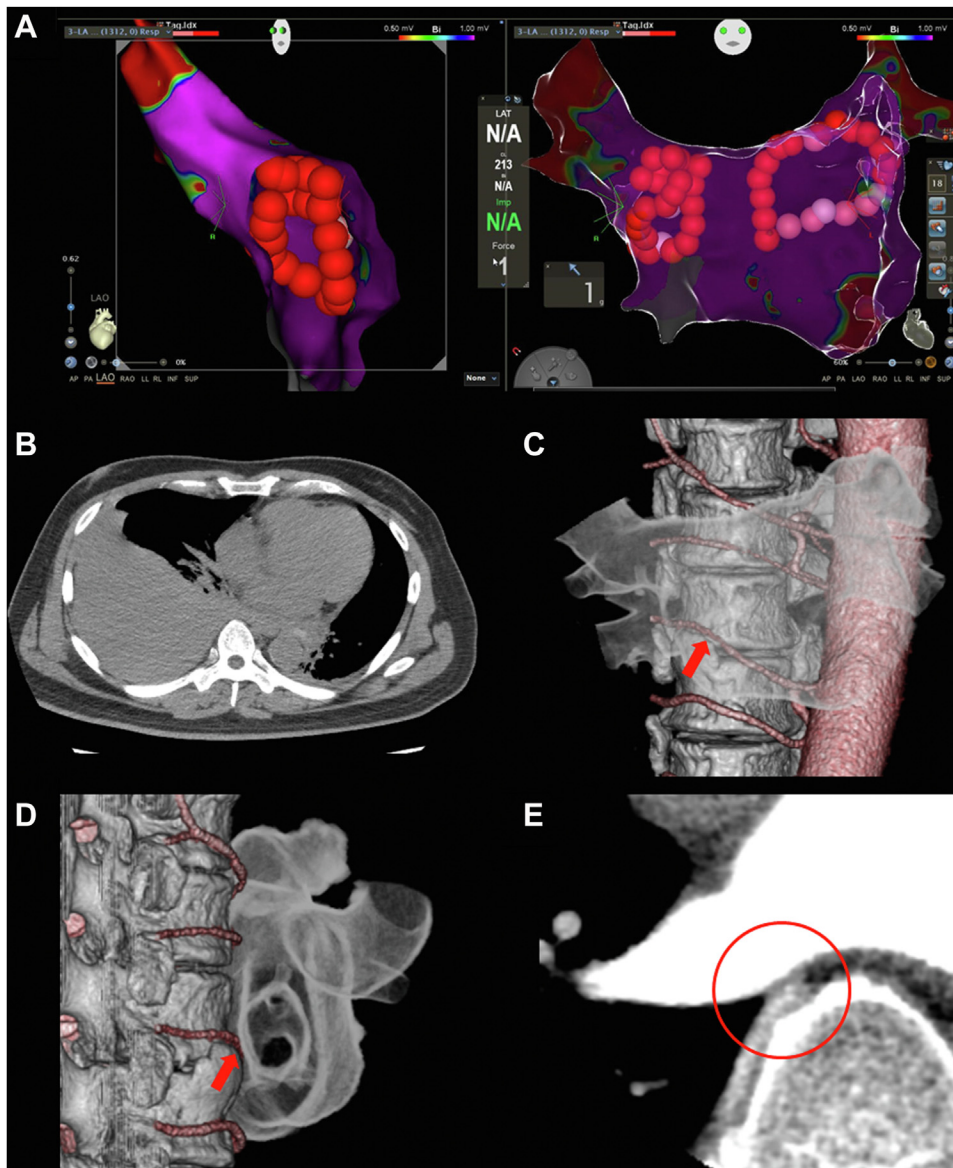


Figure 2 **A:** Voltage map with lesion tags during pulmonary vein isolation in case 2: inner view of right pulmonary vein and front view of left atrium. **B:** Noncontrast computed tomography of a massive hematoma in the right thoracic cavity. **C:** Front view of a preoperative contrast-enhanced computed tomography image. The right eighth intercostal artery branches off from the descending aorta at the level of the left atrium base and travels to the right side (red arrow). **D:** Right lateral view. The right eighth intercostal artery runs through a very narrow posterior mediastinum between the posterior wall of the left atrium and the vertebral body. **E:** Axial view. The right inferior pulmonary vein and vertebral body sandwich the eighth intercostal artery (red circle).

respectively. In particular, the point-by-point lesion parameters of the ablation for the inferior part to the posterior wall side of the right inferior PV were as follows: average contact force, 8.4 ± 1.3 g; radiofrequency power, 40 W; delivery time, 25 ± 4 seconds; and average ablation index, 462 ± 18 . The patient complained of back pain at 1 hour after the procedure. Noncontrast CT findings showed a high-density pleural effusion in the right thoracic cavity (Figure 2B), which was diagnosed as hemothorax, and emergency surgical hemostasis was attempted. During surgery in the left lateral decubitus position with right

intercostal chest opening, we identified bleeding from the right eighth ICA and ligated its origin. There was no PV or superior vena cava bleeding directly related to the ablation. Thereafter, the patient did not report any bleeding, the hemodynamics remained stable, and the hemoglobin level was retained at 12–14 g/dL without blood transfusion. Rivaroxaban (15 mg) was reinitiated on day 8, and we verified no hemorrhagic complications within 4 days after resumption of oral anticoagulant. The patient was discharged on day 12 without sequelae and has not reported any recurrence of AF during the 12-month follow-up period.

Discussion

The findings of a previous case report of right hemothorax after AF ablation suggest that the atrial septal puncture needle damaged the posterior wall of the left atrium (LA).⁴ To the best of our knowledge, this is the first report of hemothorax due to ICA injury by AF ablation.

Mechanism of the intercostal artery injury

The right ICA branches from the descending aorta and leads into the right intercostal space. Preoperative 3D CT in case 2 revealed that the injured eighth ICA had branched off from the aorta at the inferior portion of the LA, traveled to the right side of the posterior mediastinum, and was closest to the posterior wall of the LA in the right inferior PV (Figure 2C and 2D).

The mechanism of ICA injury may be that the heat energy of the radiofrequency or mechanical pressure of the ablation catheter reached the ICA through the LA wall and caused damage during PV isolation. As a result, bleeding occurred in the posterior mediastinum, thus increasing its internal pressure, perforating the parietal pleura, and spreading to the thoracic cavity.

There seem to be several factors responsible for ICA injury during AF ablation: the distance from the LA posterior wall to the ICA, the thickness of the LA wall, the location of the ICA traveling behind the LA posterior wall, the diameter of the ICA, the contact force of the ablation catheter, radiofrequency energy, or its delivery time. We used the standard contact force, radiofrequency energy, and delivery time for patients in our hospitals. The preoperative CT in case 2 revealed that the right eighth ICA was sandwiched between the posterior wall of the LA and vertebral body (Figure 2E), which may have contributed to the occurrence of an ICA injury in this case. In contrast, the right seventh ICA runs anterior to the vertebral body at the level of the right superior PV. The right superior PV is located ventral to the right inferior PV and tends to be further away from the ICA running in front of the vertebral body. Therefore, the risk of developing right seventh ICA injury during PV isolation might be lower than that of developing right eighth ICA injury.

Hemothorax occurred only on the right side in both patients. The thicknesses of the LA wall vary depending on the location; especially, the posterior wall becomes thinner in the antrum than in the center of the LA.⁵ The ICA injury appears to occur more often near the right PV, which has a thinner wall than the LA center. Another cause is that the left ICA is unlikely to contact the LA wall or left PV. The left ICA differs from the right ICA in that it branches off the descending aorta and then travels to the left intercostal space, which lies dorsal to the descending aorta. The LA is located ventral to the descending aorta, and the left ICA injury might not occur during PV isolation unless the descending aorta runs on the right side of the vertebral body.

In our study, the ICA injury occurred during PV isolation by radiofrequency ablation in both the patients. Although the

question is whether similar events will occur with cryoballoon ablation, we expect that the incidence of such events is lower with cryoballoons because local contact pressure with the atrial wall is lower with cryoballoons than with radiofrequency catheters.

Management of the intercostal injury

If an ICA injury causes hemothorax, the options for hemostasis are open surgery and coil embolization. In general, either treatment could be applicable depending on the patient's condition and the location of bleeding. In a study of 30 patients who underwent coil embolization for hemothorax caused by ICA hemorrhage, half of the patients required open or thoracoscopic surgical treatment after embolization. The mortality rate within 30 days postoperatively was as high as 23%–30.8%.^{6,7} In our study, 1 of 2 patients underwent coil embolization, which resulted in rebleeding. Ultimately, both patients underwent surgical hemostasis. In cases where the patient's condition worsens owing to persistent bleeding, it will be challenging to use the lateral decubitus position during surgery, resulting in poor vision around the posterior mediastinum where the right inferior PV and right ICA are located. Before surgery, confirmation of the origin of the bleeding by contrast-enhanced CT is not mandatory. Surgical hemostasis should be initiated as early as possible before the patient's condition worsens.

Prevention and early detection

There is a risk of ICA injury during the isolation of the right PV, where the wall is thinner from the inferior part to the posterior wall side of the right inferior PV. When contrast-enhanced CT is performed preoperatively for AF ablation, it is advisable to confirm the location between the right ICA and the LA on a 3D or axial image. The radiofrequency power, contact force, and ablation time can be adjusted if the ICA is near the right PV isolation line. However, a limitation of our study is that we cannot propose specific lesion parameter criteria that could avoid ICA injury based solely on our experience. We could detect a right hemothorax after the end of the procedure in both cases but not during the procedure. Early detection of pleural effusion by fluoroscopy or echocardiography during the procedure is an optimal approach.

Conclusion

Bleeding from the ICA rarely occurs during AF ablation but can lead to severe hemothorax. Contrast-enhanced CT can precisely provide the location of the ICA behind the LA posterior wall and be useful to assess the risk of this complication during the ablation. If hemothorax occurs, we should consider surgical treatment as early as possible.

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