

RHYTHM DISORDERS AND ELECTROPHYSIOLOGY

CLINICAL CASE

Acute Pericarditis After Pulsed Field Ablation for Atrial Fibrillation



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ABSTRACT

BACKGROUND Pulsed field ablation (PFA) is a novel nonthermal ablation technique for atrial fibrillation, offering myocardial selectivity and a favorable safety profile. However, postprocedural pericarditis remains rarely reported.

CASE SUMMARY A 62-year-old man with paroxysmal atrial fibrillation underwent PFA with intracardiac echocardiography. Ablation was delivered at 2.0 kV, and postprocedural intracardiac echocardiography showed no pericardial effusion. Twelve hours later, the patient developed pleuritic chest pain. Electrocardiography showed PR-segment depression and diffuse ST-segment elevation. Transthoracic echocardiography revealed a 3-mm pericardial effusion, with elevated inflammatory biomarkers. Acute pericarditis was diagnosed, and the patient was treated with hydrocortisone, ibuprofen, and colchicine, resulting in symptom resolution within 4 weeks.

DISCUSSION Despite the nonthermal mechanism of PFA, acute pericarditis may occur as an early complication. Indirect myocardial inflammation and immune-mediated responses may contribute. Early recognition enables timely management.

TAKE-HOME MESSAGES Acute pericarditis can occur after PFA. Prompt anti-inflammatory therapy is essential for optimal outcomes. (JACC Case Rep. 2025;30:104811) © 2025 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

Pulmonary vein isolation (PVI) is considered an effective rhythm control treatment for atrial fibrillation (AF), and its use is increasing exponentially. Pulsed field ablation (PFA) has gained prominence owing to its selective electroporation mechanism, minimizing collateral damage to adjacent structures. Although PFA has a low complication profile,¹ postprocedural pericarditis after PFA remains rarely reported. We report a case of acute

TAKE-HOME MESSAGES

- Acute pericarditis is a rare but possible early complication following pulsed field ablation for atrial fibrillation.
- Early recognition of symptoms and timely initiation of the anti-inflammatory therapy are essential to ensure favorable clinical outcomes.

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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](#).

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ABBREVIATIONS AND ACRONYMS

AF = atrial fibrillation
CK-MB = creatine kinase-muscle brain
ECG = electrocardiography
hsTnI = high-sensitivity troponin I
ICE = intracardiac echocardiography
LA = left atrium
PFA = pulsed field ablation
PV = pulmonary vein
TTE = transthoracic echocardiography

pericarditis immediately after PFA, emphasizing its clinical course, diagnosis, and management.

HISTORY OF PRESENTATION

A 62-year-old man (weight 52 kg, height 161 cm) with drug-refractory paroxysmal AF presented for rhythm control with PFA. Transthoracic echocardiography (TTE) revealed an ejection fraction of 65%, with a left atrial dimension of 31 mm and volume index of 28.5 mL/m². The initial electrocardiogram (ECG) showed a normal sinus rhythm, and coronary computed tomography angiography showed no significant stenosis, indicating normal coronary arteries (Figure 1).

We conducted PFA using a 31-mm Farawave catheter (Farastar) under deep sedation with intravenous propofol and fentanyl. Anticoagulation with 60 mg edoxaban was uninterrupted, and activated clotting time was maintained between 300 and 350 seconds during the procedure. For each pulmonary vein

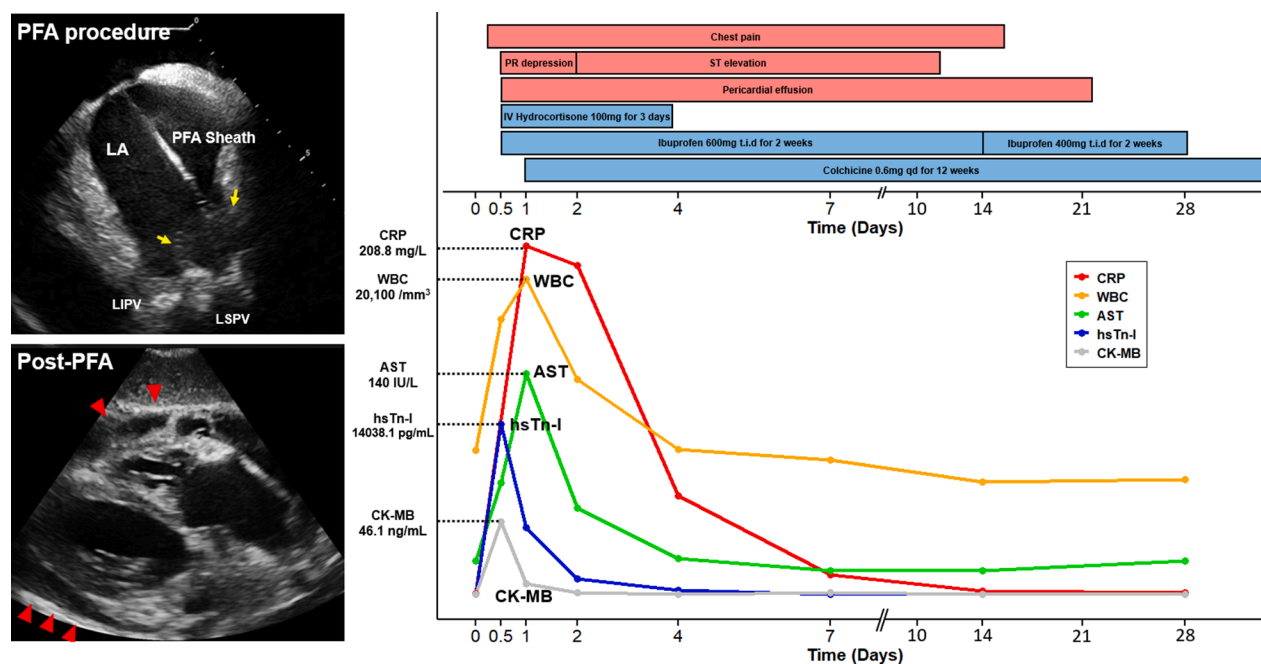
(PV)—left superior PV, left inferior PV, right superior PV, and right inferior PV—pulses were delivered twice at the ostial level in a basket-shaped configuration (Figures 2A to 2D) and twice at the antral level in a flower-shaped configuration (Figures 2E to 2H), resulting in a total of 32 ablations. Each ablation consisted of 5 pulse trains with a biphasic voltage of 2.0 kV within 2.5 second. Intracardiac echocardiography was used to confirm adequate contact of the Farawave catheter at each PV. Before retracting the catheter and sheath from the left atrium, we rechecked PVI and ascertained no procedural complications, including pericardial effusion using intracardiac echocardiography.

Twelve hours post-procedure, the patient developed mild pleuritic chest pain that worsened in the supine position and improved by leaning forward.

PAST MEDICAL HISTORY

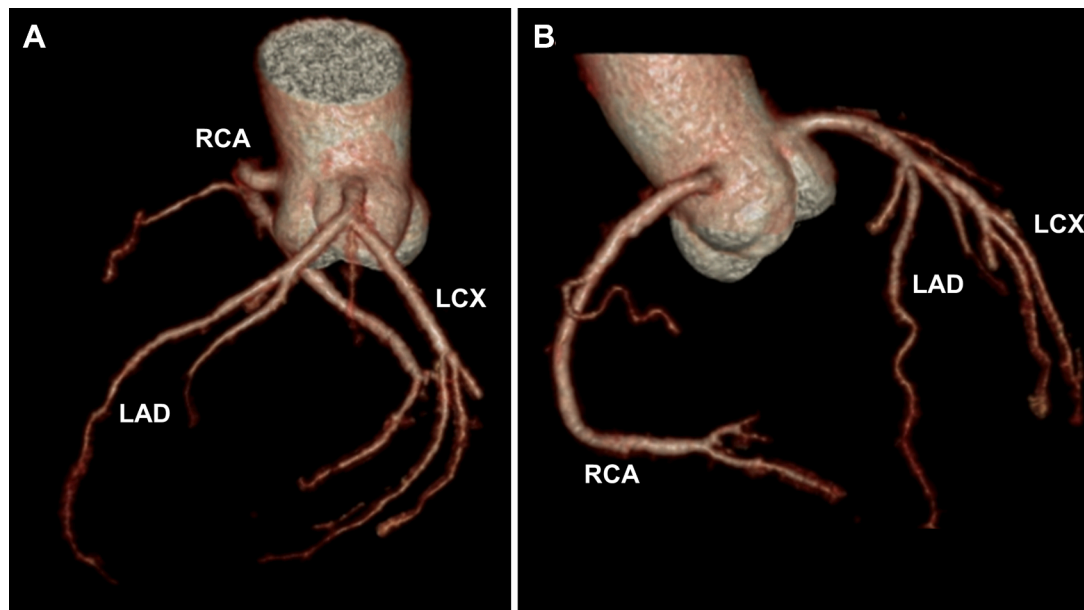
The patient had a history of type 2 diabetes mellitus managed with oral metformin. He had no known history of pericardial disease, coronary artery disease, or autoimmune disorders.

VISUAL SUMMARY Acute Pericarditis Following Pulsed Field Ablation



(Left top) Intraprocedural intracardiac echocardiography during PFA shows catheter positioning in the left atrium. The catheter is in contact with the LSPV (yellow arrow). (Left bottom) Postprocedural transthoracic echocardiography reveals pericardial effusion with inflammatory debris (red arrows). (Right) Clinical course timeline detailing onset of chest pain, electrocardiographic changes (PR depression, ST-segment elevation), pericardial effusion, and anti-inflammatory treatment (hydrocortisone, ibuprofen, colchicine) and serial inflammatory biomarker trends. AST = aspartate aminotransferase; CK-MB = creatine kinase-muscle brain; CRP = C-reactive protein; hsTn-I = high-sensitivity troponin I; LA = left atrium; LIPV = left inferior pulmonary vein; LSPV = left superior pulmonary vein; PFA = pulsed field ablation; WBC = white blood cell.

FIGURE 1 Coronary Computed Tomography Angiography With 3-Dimensional Reconstruction



Three-dimensional reconstruction of coronary computed tomography angiography shows no significant coronary artery stenosis, consistent with normal coronary anatomy: (A) anteroposterior cranial view. (B) Left anterior oblique view. LAD = left anterior descending artery; LCX = left circumflex artery; RCA = right coronary artery.

DIFFERENTIAL DIAGNOSIS

The initial differential diagnosis of postprocedural pleuritic chest pain included acute pericarditis, myocardial ischemia, and cardiac tamponade.

INVESTIGATIONS

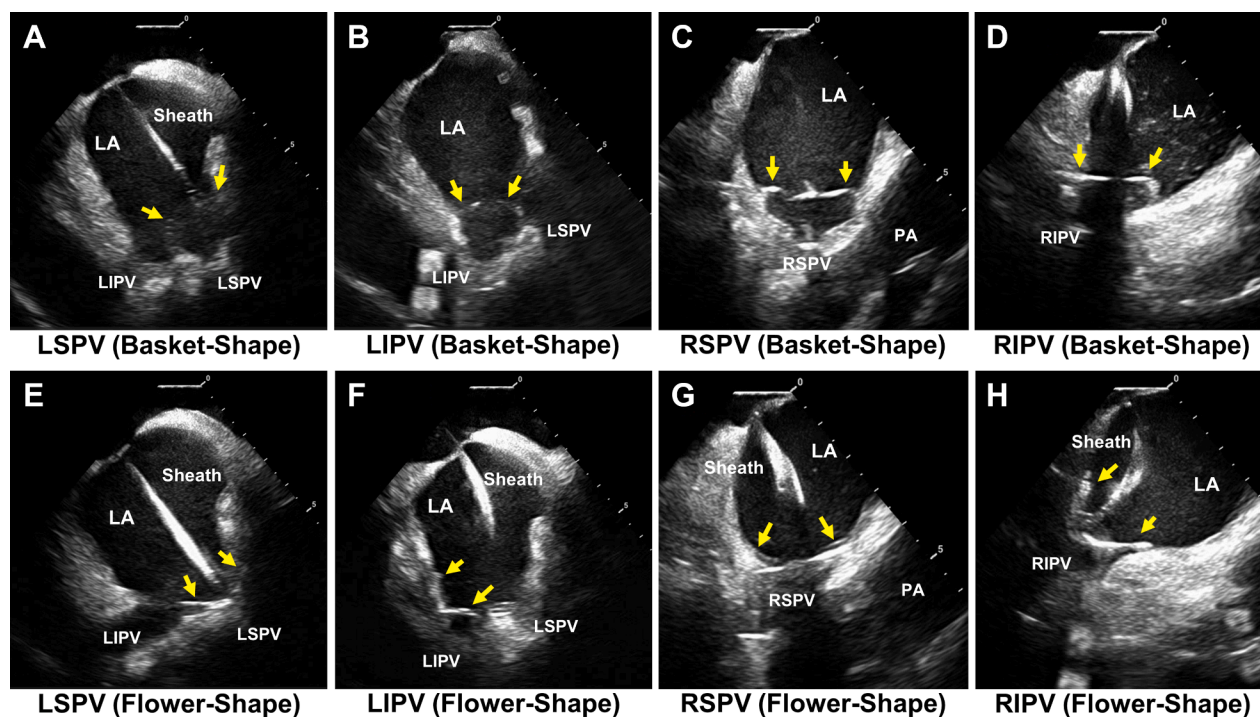
Twelve hours post-ablation, ECG revealed mild PR-segment depression (Figure 3A), and follow-up ECG at 24 hours showed ST-segment elevation >2 mm in both limb and precordial leads (Figure 3B). TTE demonstrated a newly developed 3-mm pericardial effusion (Figure 4A), which slightly increased by day 10 with inflammatory debris in the pericardial space (Figure 4B). Laboratory testing revealed elevated white blood cell, high-sensitivity troponin I, creatine kinase-muscle brain (CK-MB), aspartate aminotransferase, and C-reactive protein levels (Figure 5). There was no evidence of hemolysis or acute kidney injury: serum creatinine and lactate dehydrogenase levels remained within normal limits, and no hemoglobin drop or macroscopic hematuria was observed.

MANAGEMENT

The patient was diagnosed with acute pericarditis and initiated on intravenous hydrocortisone

(100 mg/day), which was continued for 3 days and discontinued after inflammatory markers began to normalize (Figure 5). Given the marked inflammatory response and the very rapid deterioration of symptoms, short-term corticosteroid therapy was used in conjunction with ibuprofen. Oral ibuprofen was prescribed at 600 mg 3 times daily for 2 weeks, then tapered to 400 mg 3 times daily for another 2 weeks. Twenty-four hours after the procedure, the patient experienced worsening chest pain. ECG demonstrated ST-segment elevation >2 mm in both limb and precordial leads (Figure 3B), and laboratory testing showed further increases in cardiac and inflammatory biomarkers (Figure 5). In response, colchicine (0.6 mg once daily) was initiated on post-procedure day 2 and continued for 12 weeks. This anti-inflammatory regimen resulted in progressive symptom improvement and biomarker stabilization.

The patient was discharged on postprocedure day 3 with improving symptoms. At 10-day follow-up, TTE revealed slightly increased pericardial effusion with inflammation debris in the pericardial space (Figure 4B). ECG abnormalities returned to normal values at 3 weeks after the procedure (Figure 3C). Four-week follow-up TTE demonstrated no pericardial effusion (Figure 4C), and cardiac biomarkers, WBC = white blood cell, aspartate aminotransferase,

FIGURE 2 Pulmonary Vein Ablation Sites With Pulsed Field Ablation Confirmed Using Intracardiac Echocardiography

Antral ablation using a flower-shaped configuration at each pulmonary vein, confirmed by intracardiac echocardiography (ICE) (yellow arrow): (A) LSPV, (B) LIPV, (C) RSPV, (D) RIPV. Ostial ablation using a basket-shaped configuration at the same pulmonary veins, also confirmed by ICE (yellow arrow): (E) LSPV, (F) LIPV, (G) RSPV, (H) RIPV. ICE = intracardiac echocardiography; LA = left atrium; LIPV = left inferior pulmonary vein; LSPV = left superior pulmonary vein; PA = pulmonary artery; RIPV = right inferior pulmonary vein; RSPV = right superior pulmonary vein.

and C-reactive protein levels returned to normal (Figure 5).

OUTCOME AND FOLLOW-UP

At 3-month follow-up, the patient remained asymptomatic without recurrence of pericarditis. He expressed satisfaction with symptom resolution and AF ablation success despite the initial concern about early postprocedural chest pain.

DISCUSSION

INCIDENCE OF PERICARDITIS AFTER PFA. Pericarditis is a recognized complication of thermal ablation techniques, with reported incidence rates varying across studies. Yadav et al² reported an incidence of 3.7% for acute pericarditis after radiofrequency catheter ablation for AF. Similarly, Mugnai et al³ observed a 4% incidence of acute pericarditis after cryoballoon ablation for AF.

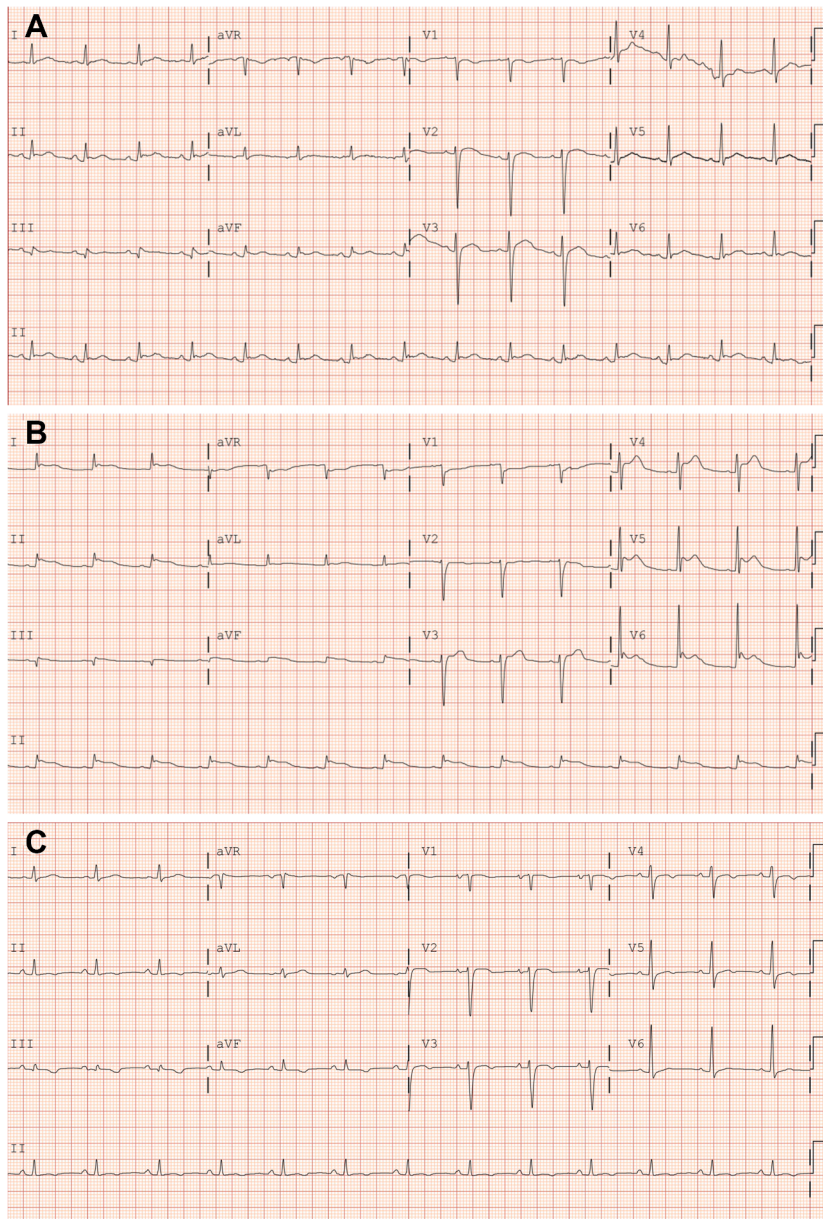
PFA, because of its nonthermal nature and cardiomyocyte selectivity, was initially thought to have a very low risk of pericardial complications.

The MANIFEST-PF (Multi-National Survey on the Methods, Efficacy, and Safety on the Post-Approval Clinical Use of Pulsed Field Ablation) registry,¹ which included 1,568 patients, reported only 1 case (0.06%) of acute pericarditis, suggesting that it remains a rare but possible complication. Similarly, the ADVENT trial,⁴ comparing PFA with conventional thermal ablation, observed acute pericarditis in 1 patient (0.3%), further supporting the notion that while PFA may reduce the procedure-related risks, pericarditis remains a potential complication.

Although cases of acute pericarditis have not been comprehensively described in the MANIFEST-PF registry, one case of acute pericarditis was treated conservatively. The ADVENT trial also reported a single case, but the specific diagnostic and therapeutic strategy was not disclosed. These limited data highlight the need for detailed case-based management strategy for acute pericarditis just after PFA, as demonstrated in the present report.

MECHANISMS OF PERICARDITIS AFTER PFA. One of the mechanisms is indirect pericardial injury. PFA is

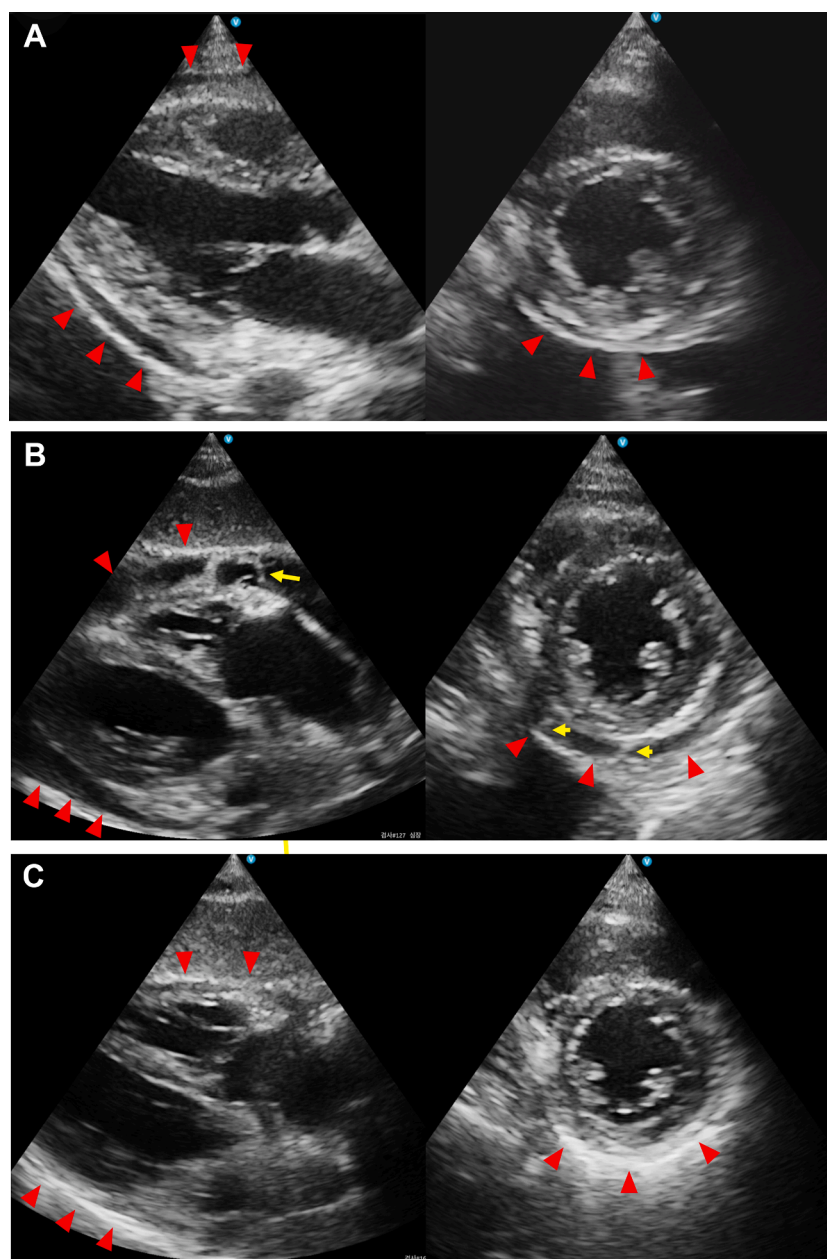
FIGURE 3 Electrocardiographic Changes in Post-Pulsed Field Ablation Pericarditis



(A) Electrocardiogram (ECG) at 12 hours after the procedure showing mild PR depression. (B) ECG at 24 hours after the procedure showing an ST-segment elevation >2 mm in limb and precordial leads. (C) ECG at 3 weeks after the procedure showing normalization of ST-segment elevation.

characterized by cardiomyocyte selectivity. The induced localized myocardial injury may trigger an inflammatory response, which in some cases could extend to the pericardium, potentially resulting in pericarditis.⁵ The pericardium consists of 2 structurally distinct layers, each with unique properties that may influence its response to PFA. The fibrous

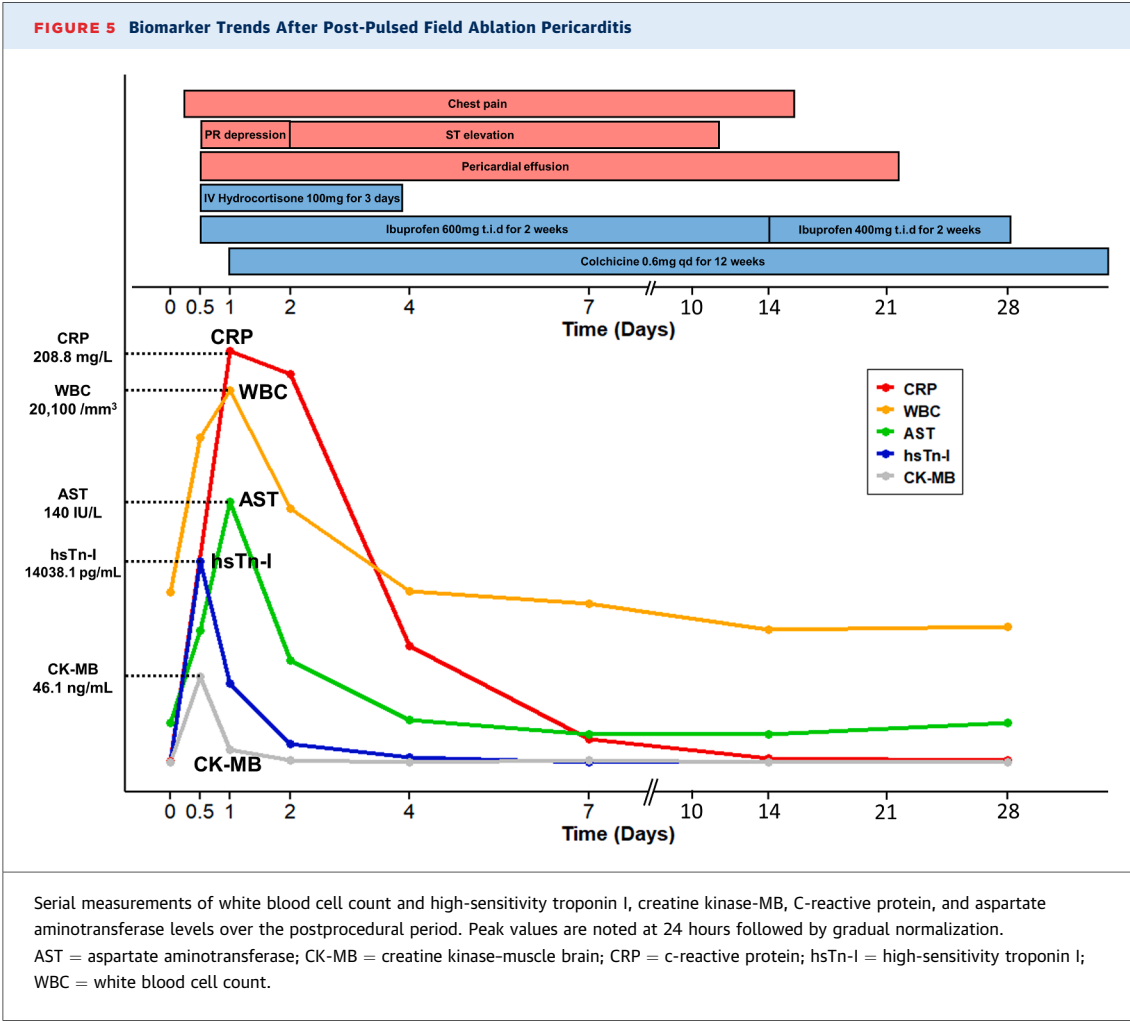
pericardium, composed of dense connective tissue, serves as a protective outer layer with high electrical resistance, making it less susceptible to the direct effects of PFA. In contrast, the serous pericardium, also known as the visceral pericardium or epicardium, is lined with mesothelial cells. When exposed to inflammatory stimuli, this layer may become

FIGURE 4 Transthoracic Echocardiographic Changes in Post-Pulsed Field Ablation Pericarditis

(A) Transthoracic echocardiography (TTE) at 12 hours after the procedure showing pericardial effusion of 3 mm (red arrow). (B) TTE at 10 days after the procedure revealed slightly increased pericardial fluid effusion of 5 mm (red arrow) with inflammatory debris in the pericardial space (yellow arrow). (C) TTE at 4 weeks after the procedure showing no pericardial effusion (red arrow).

irritated, leading to pericardial effusion and pericarditis. Pericardial inflammation after PFA occurs not because of direct electroporation of the pericardium but rather as a secondary response to myocardial injury and subsequent inflammatory response.⁶

Another plausible mechanism for PFA-associated pericarditis is an immune-mediated response, similar to postcardiac injury syndrome (PCIS). PCIS includes conditions similar to pericarditis that arise after cardiac injury, including interventions such as



pacemaker lead insertions, percutaneous coronary interventions, and radiofrequency ablations.⁷ Specifically, tissue damage results in the accumulation of debris and blood in the pericardium, which can trigger immune responses that maintain inflammation in susceptible individuals. This process may involve the formation of immune complexes and activation of cell-mediated immunity, contributing to the development of pericarditis.

OPTIMIZATION OF VOLTAGE SETTINGS IN PFA.

Voltage outputs from several PFA generators typically range from 1.5 kV to 2.0 kV for PVI. Lowering the voltage below 1.5 kV has been explored to reduce the risk of collateral injury and hemolysis of red blood cells; however, inadequate energy application may lead to nontransmural damage or reversible electroporation of cardiomyocytes, increasing the risk of PV reconnection and AF recurrence. A comprehensive dose-response study of PFA using a focal ablation

catheter revealed that while lower-voltage settings might reduce collateral damage, they could also result in insufficient lesion formation, thereby compromising the efficacy of the ablation.⁸ Furthermore, the voltage settings of each PFA generator have been shown to effectively achieve durable PVI with favorable safety profiles.⁴ However, the energy settings are not individualized. The patient in the present study had a small stature and has a very small left atrium size (31 mm). Therefore, an energy of 2.0 kV may be extremely high to perform PVI without collateral damage in some conditions. Further dose-response studies of PFA with multielectrode catheters are needed to clarify unresolved issues.

PHARMACOLOGIC MANAGEMENT OF POST-PFA PERICARDITIS.

Nonsteroidal anti-inflammatory drugs (NSAIDs) are widely recommended as the first-line treatment for acute pericarditis owing to their anti-inflammatory properties and ability to relieve pain.

NSAID therapy, particularly when combined with colchicine, significantly reduces pericarditis recurrence compared to NSAIDs alone. A meta-analysis found that colchicine in combination with an NSAID lowered the recurrence rate of pericarditis and improved symptom resolution.⁹ Given the suspected immune-mediated mechanism of acute pericarditis after PFA, the use of NSAIDs in combination with colchicine may be more effective in resolving inflammation and managing symptoms than NSAIDs alone.

Corticosteroids are typically reserved for refractory pericarditis or cases where NSAIDs and colchicine are contraindicated. Although corticosteroids provide rapid symptom relief, studies have shown that their use is associated with higher recurrence rates and may prolong the disease course by impairing viral clearance and altering immune response mechanisms.¹⁰ Although corticosteroids are generally reserved for refractory or NSAID-intolerant pericarditis, we initiated a short 3-day course of intravenous hydrocortisone early in the clinical course of the patient with a marked inflammatory response and increasingly severe chest pain. This decision was based on clinical judgment given the severity of symptoms and biomarker elevation. While corticosteroids have been associated with higher recurrence in viral or idiopathic pericarditis, they may be beneficial in immune-mediated cases such as PCIS, which shares mechanistic features with pericarditis following ablation.¹⁰ In cases of severe inflammatory response after PFA, a short-term (3-day) administration of corticosteroids may be considered

to reduce excessive inflammation and alleviate symptoms.

CONCLUSIONS

This case underscores that acute pericarditis can occur after PFA. Recognizing postprocedural chest pain patterns and integrating multimodal diagnostic tools are critical for timely diagnosis and prompt therapeutic intervention. Unlike previous registry or clinical trial reports that only briefly mention pericarditis without clinical details, this case provides a comprehensive description of the diagnostic pathway, inflammatory marker changes, and effective management strategies. It also underscores the importance of individualized energy titration in patients at high risk of immune-mediated inflammatory complications. Further studies are warranted to elucidate the underlying pathophysiology of PFA-induced pericarditis and refine risk-mitigation strategies.

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

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