

Case Report

Acute post-ablation pericarditis with insights into clinical trajectory, temporal electrocardiogram and biochemical trends after pulsed field ablation for atrial fibrillation

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Case report

A 66-year-old gentleman was reviewed in the outpatient cardiology clinic for symptomatic paroxysmal Atrial Fibrillation (AF) and atrial flutter, first diagnosed in August 2025. His significant past medical history includes diabetes mellitus, hypertension, hyperlipidaemia, chronic kidney disease and ischemic heart disease requiring percutaneous coronary intervention to the first obtuse marginal coronary artery. Transthoracic echocardiogram in 2025 demonstrated left ventricular ejection fraction of 50% and normal left atrial volume of 64ml (volume index of 32.8 ml/m²).



Figure 1: Baseline 12-lead electrocardiogram pre-ablation. Atrial fibrillation was intermittently seen organising into atrial flutter.

He underwent elective Pulsed Field Ablation (PFA) that included Pulmonary Vein Isolation (PVI), left atrial roof and Posterior Wall Ablation (PWA), and Cavo-Tricuspid Isthmus (CTI) ablation using the Boston Scientific Farapulse PFA system on 2nd October 2025. The procedure was performed with a 3-dimensional electro-anatomical mapping system (Abbott Ensite X EP System), intracardiac echocardiogram, and fluoroscopy. Total ablation lesions (27 Balloon (B) and 55 Flower (F) applications) were delivered, comprising 4B 8F to the left superior pulmonary vein, 4B 8F to the left inferior pulmonary vein, 4F to the left carina, 4B 4F to the right superior pulmonary vein, 4B 10F to the right inferior pulmonary vein, 4F to the right carina, 17F to the left atrial roof and posterior wall region, and 11B to CTI region. Prior to CTI ablation, intravenous glyceryl trinitrate 1 mg

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slow bolus was administered to avoid the potential adverse effect of right coronary artery spasm. Empirical PWA and CTI ablation were performed due to documented atrial flutter. Mitral Isthmus (MI) line ablation was not performed due to increased tissue thickness seen in that region on ICE imaging and anticipated difficulty in achieving blocked MI line using PFA alone. The total procedural ablation time was 44 minutes, and the left atrial dwell time was 85 minutes.

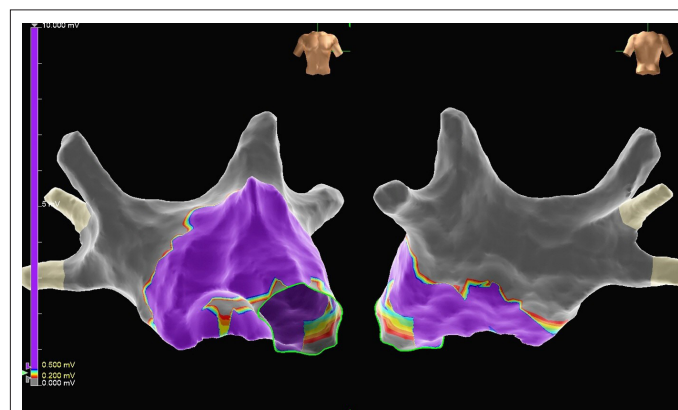


Figure 2: Post-ablation re-map showed successful pulmonary vein isolation, left atrial roof and posterior wall ablation.

Two hours post-procedure, he developed acute onset central chest pain and dyspnoea. Chest pain was influenced by positional change, worsening with supine position and during deep inspiration. He was haemodynamically stable (temperature 36.6°C, blood pressure 116/69 mmHg, pulse rate 72 regular, oxygen saturation 99% on room air). Physical examination was unremarkable. No adventitious sounds were heard on auscultation of the heart and lungs. Jugular venous pressure was not elevated. There was no obvious pericardial rub.

The serial 12-lead electrocardiograms in the first 24 hours demonstrated sinus rhythm with minimal ST segment or T wave changes.

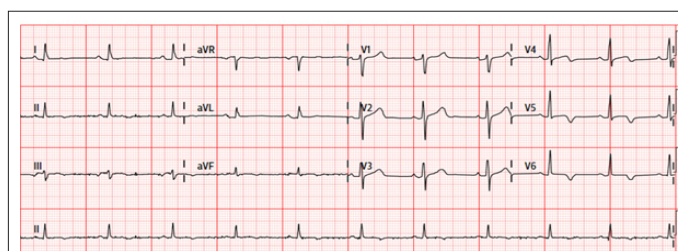


Figure 3: Serial 12-lead electrocardiograms performed within 24 hours post-ablation. There were no acute ST segment or T wave changes seen as compared to baseline.



Figure 4: Small circumferential 7mm pericardial effusion demonstrated on transthoracic echocardiogram.

Blood tests showed raised Troponin T 8689 ng/L, Creatinine Kinase-Myocardial Band (CKMB) 73.6 µg/L, and Creatinine Kinase (CK) 1036U/L. Leukocyte count and C-Reactive Protein (CRP) levels were elevated at 23.4 x10⁹/L and 171 mg/L respectively.

Chest radiograph was within normal limits.

A bedside transthoracic echocardiogram showed the presence of small pericardial effusion.

Clinical impression was likely acute post-ablation pericarditis, and he was promptly commenced on oral Ibuprofen 600 mg 3 times per day and oral Colchicine 500mcg once daily (dose reduced due to chronic kidney disease). Other differential diagnoses include acute coronary spasm and cardiac perforation. The nature of the chest pain, late peaking in chest pain severity, along with minimal changes in serial electrocardiograms within the first 24 hours supports a non-coronary event. Cardiac biomarkers, including Troponin T, were not helpful to exclude coronary artery spasm in this post-ablation setting as they were expected to be markedly elevated following PFA. Cardiac perforation was excluded in the absence of worsening haemodynamic, significant pericardial effusion, or tamponade.

Despite the initial treatment, his chest pain worsened on post-procedural day 2. Repeated electrocardiograms 24-48 hours post ablation now showed clear and typical findings of acute pericarditis. There was diffuse concave ST segment elevation accompanied with prominent J point elevation and S wave in precordial leads; the lack of terminal QRS distortion favoured a non-ischaemic aetiology, making coronary artery spasm less likely [1]. Pronounced reciprocal PR segment elevation and ST segment depression were observed in lead aVR.

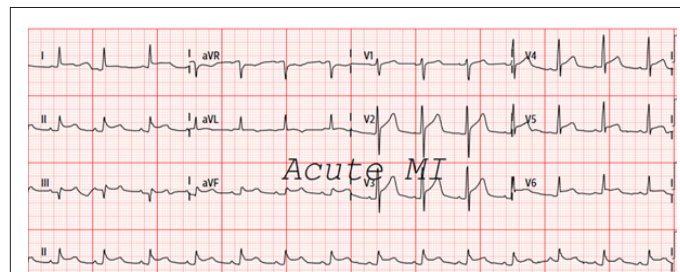


Figure 5: Repeated electrocardiogram 24-48 hours post-ablation showing diffuse concave ST segment elevation with J point elevation and S wave in precordial leads. This was mis-labelled as acute myocardial infarction by electrocardiogram machine algorithm.

Based on 2025 Concise Clinical Guidance (CCG): An ACC Expert Consensus Statement on the Diagnosis and Management of Pericarditis [2], our patient fulfilled definite diagnosis of acute pericarditis given the presence of pleuritic chest pain, elevated inflammatory biomarkers, diffuse ST segment elevation on electrocardiogram, and new pericardial effusion on transthoracic echocardiogram. Of note, typical electrocardiogram changes were not apparent in the first 24 hours.

Despite the use of first-line anti-inflammatory dual therapy with Non-Steroidal Anti-Inflammatory Drug (NSAID) and colchicine, he experienced worsening chest pain and dyspnoea, and peak in symptom severity beyond the first 24 hours from ablation. Intravenous hydrocortisone 100 mg once dose was added given the severity of his symptoms, which abated almost immediately after. At 36 hours post-procedure, he developed a transient fever of 38.6°C. We elected not to commence any

antibiotics and continue to observe the fever trend, which lysed over the next 12 hours. Intravenous hydrocortisone 100mg once daily was continued for another 2 more days, in addition to oral Ibuprofen and oral Colchicine.

The trend of biomarkers over nearly 90 hours was illustrated in (Table 1). His cardiac biomarkers, Creatinine Kinase-MB (CK-MB), returned to normal by post-ablation day 4, while both Troponin-T and Creatinine Kinase (CK) levels decreased fivefold. The leukocyte count peaked at 36 hours post-procedure and halved by day 4. Similarly, C-Reactive Protein (CRP) level reached its highest point at 60 hours post-ablation with ongoing pericarditis treatment, before it started to fall by day 4.

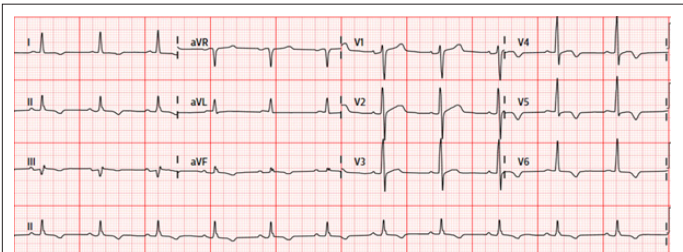


Figure 6: Repeated electrocardiogram during outpatient clinic review on post-ablation day 19. There was resolution of diffuse ST segment elevation and new T wave inversion in the inferior leads.

Table 1: Trend of Biomarkers during the patient’s admission.

	Baseline	Hours post-procedure					19 days post-procedure
		2	30	36	60	84	
Troponin T (ng/L) (<= 29 ng/L)	28-29	8689	7325	8354	4333	1766	-
CK (U/L) (40-320 U/L)	-	1036	779	675	387	259	-
CK-MB (ug/L) (<= 4.9 ug/L)	-	73.6	41.8	25.4	5.7	3.2	2.1
Leukocyte Count (x10 ⁹ /L) (4-10 x10 ⁹ /L)	6-8	23.40	-	20.22	18.40	12.71	6.73
CRP (mg/L) (<= 4.9 mg/L)	-	18.3	55.0	90.4	171.0	107.0	< 0.06

CK: Creatinine Kinase; CK-MB: Creatinine Kinase-MB; CRP: C-Reactive Protein.

Our patient was discharged well on post-ablation day 4 with instructions to continue oral Ibuprofen for 1 week and oral Colchicine for 3 months in duration. No oral corticosteroids were prescribed on discharge. He was reviewed in the Cardiology outpatient clinic on post-ablation day 19 and was found to have complete resolution of symptoms. (Table 1) showed that his leukocyte count, CRP level and cardiac enzymes had largely normalised. Repeat electrocardiogram showed resolution of diffuse ST segment elevation and new T wave inversion in the inferior leads, reflecting expected temporal electrocardiogram changes from stage 2 to stage 3 then. He remained stable, asymptomatic and in sinus rhythm during his last clinic consultation on 20th March 2026, approximately over 5 months from his AF PFA procedure. There was no recurrence of pericarditis noted after resolution of the initial episode.

Discussion/Conclusion

From this case report, key observations include the following:

- 1. Acute post-ablation pericarditis needs to be promptly considered in the presence of chest pain following AF PFA procedure. Typical 12-lead electrocardiogram pericarditis changes tend to lag behind clinical presentation and may not be apparent in the first 24 hours.
- 2. The 12-lead electrocardiogram machine algorithm has high sensitivity in the detection of acute myocardial infarction as seen in our case, but such electrocardiograms may need to be further verified by physicians for accurate interpretation within the clinical context. Other differentials, e.g., pericarditis or coronary artery spasm, are more likely to account for acute post-ablation electrocardiogram changes.

3. Current guidelines are unclear regarding the next line of pharmacotherapy for patients not responding to contemporary first-line pharmacotherapy. We supported the use of a short course of intravenous hydrocortisone treatment for post-ablation pericarditis with favourable response seen in our patient.

Acute pericarditis has been well characterised following thermal AF ablation [3,4]. However, there is limited data on the incidence of acute post-ablation pericarditis in patients who underwent PFA. Ise egger et al. reported that multiple registries, e.g., MANIFEST-AF, EU-PORIA and ADVENT trials, have previously quoted an incidence of between 0.06 - 0.3% in their cohorts [5]. However, little detail was provided on how these cases were diagnosed and managed. Despite being myocyte-selective, the occurrence of post-ablation pericarditis suggests that PFA ablation may still be able to generate a significant inflammatory response in mesothelial cells and connective tissues within pericardium, albeit at a lower incidence compared with AF ablation using thermal sources. The specific effects of PFA on mesothelial cells are not adequately evaluated and may possibly result from direct tissue insult or indirect near-field tissue heating secondary to electroporation.

Jung et al. hypothesised that the mechanism of post-ablation pericarditis in PFA could likely be immune-mediated inflammation, involving cell-mediated immune responses to accumulated by-products from tissue injury within the pericardial space [6]. An inflammatory phenotype was demonstrated in our patient by the presence of fever, increased leukocyte count and CRP level. Looking at electroporation biophysics [7] and pericarditis pathophysiology [8], both pericardial inflammation with PFA and acute pericarditis may share common mechanisms involving release of Damage-Associated Molecular Patterns

(DAMP) and generating of Reactive Oxygen Species (ROS). These stimuli then trigger a response in inflammasomes, macromolecular cellular structures playing key roles in innate immunity, resulting in activation of inflammatory cytokines, such as interleukin-1 β involved in acute pericarditis.

In our patient whose clinical trajectory was initially refractory to first-line therapy using non-steroidal anti-inflammatory drugs and colchicine, the rapid clinical improvement and symptom resolution following intravenous corticosteroids may also highlight the probable key role of immune constitution in the pathophysiology contributing to post-ablation pericarditis. Hence, there may be a role to consider short course of intravenous corticosteroids for the treatment of this unique entity.

From our experience, use of the Farapulse PFA system, which is a relatively larger footprint Penta spline catheter, in combination with an extensive ablation lesion set, may have led to the incidence of post-ablation pericarditis. Underlying postulated mechanisms include inadvertent mechanical micro-injury within a fairly normal sized left atrium, near-field tissue heating, and immune-mediated inflammatory responses similar to a Dressler-like mechanism extending to the pericardium. These factors may result in the occurrence of acute post-ablation pericarditis in PFA, with the possibility of added risks when PWA is performed. The NEMESIS-PFA study provided further insights to cardiac injury pattern and biomarker assessment between PFA and radiofrequency ablation (RFA) [9]. There is greater extent of myocardial injury as shown by the significantly larger increase in post-procedural Troponin T level with PFA in comparison to RFA. The study revealed a dose-response relationship with the number of PFA applications and degree of Troponin T level rise [9]. The largest post-procedural Troponin T level was found in the Penta spline catheter design among all PFA systems.

Pérez-Pinzón et al. showed that the occurrence of post-ablation pericarditis can also be related to PFA catheter design [10]. The large surface area of Penta spline catheters facilitates rapid and efficient ablation. On the other hand, more PFA applications may be necessary to achieve electrical isolation in certain left atrial regions as there can be difficulty in obtaining lesion apposition using such catheters [10]. The rapid delivery rate of PFA applications, in addition to increased number of applications and lack of good tissue contact at times, may increase the risk of developing post-ablation pericarditis. Conversely, Izba et al. reported that higher numbers of PFA applications were not associated with acute post-ablation pericarditis, but paradoxically, shorter procedural times were found to be related [11]. Nonetheless, continued monitoring for pericarditis and other complications is considered good clinical practice with every new and pre-existing PFA system.

Acute pericarditis is an uncommon but potentially underdiagnosed complication following PFA. There may be a time delay in the development of typical electrocardiogram changes. This case highlights the importance of having an early clinical suspicion for post-ablation pericarditis. Understanding the temporal electrocardiogram changes and biochemical trends would help physicians in deriving the right diagnosis. An immune-mediated inflammatory response could have played a significant role in the occurrence of post-ablation pericarditis.

In severe, refractory cases of post-ablation pericarditis, similar to what our patient experienced, a short course of intravenous corticosteroids may be considered. Further research is crucial to elucidate the true incidence of post-ablation pericarditis in PFA, underlying mechanisms, risk factors, differences across all PFA systems, and optimal treatment approach.

Declarations

Conflict of interest: XM Pung, TS Eric Lim, KL Ho, TT Daniel Chong and CK Ching received honoraria from Boston Scientific for talks unrelated to this manuscript. JX Ng declare no conflicts of interest.

All authors have no disclosure to declare.

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