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# Steroid Responsive Pericardial Effusion after Percutaneous Epicardial Closure of the Left Atrial Appendage

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**Abstract**

Atrial Fibrillation (AF) increases the risk of embolic Cerebrovascular Accidents (CVA) with the thrombus predominately originating in the Left Atrial Appendage (LAA). In patients unable to take oral anticoagulation, closure or exclusion of the LAA can be performed to mitigate the risk of stroke. We present a case of a patient who underwent LAA closure with an epicardial suture delivery device, and developed a late post procedure pericardial effusion despite perioperative anti-inflammatory therapy. This was successfully treated conservatively with oral steroids, negating the need for further invasive therapy with pericardiocentesis.

**Keywords**: Atrial fibrillation; Anticoagulation; Left atrial appendage Closure; Pericardial effusion

## Introduction

Atrial Fibrillation (AF) has an estimated prevalence of 1.5-2% currently [1], and it is expected to increase from a prevalence of 5.2 million cases in 2010 to an estimated 12.1 million cases in 2030 [2]. AF has been associated with an increased risk of embolic Cerebrovascular Accidents (CVA). In patients with non-valvular AF, the risk of having an embolic CVA is increased 5.6 fold. This incidence increases with age and with increasing comorbidities [3,4]. Warfarin, a vitamin K antagonist and, more recently, Novel Oral Anticoagulants (NOACs) are prescribed to mitigate the risk of CVA in patients with AF [5]. Warfarin has been shown to reduce the risk of embolic CVA in AF patients by up to 62% [6]. Stroke prophylaxis with NOACs has shown to be non-inferior and, in some cases, superior to warfarin therapy in this patient population. Major bleeding complications are lower with the NOACs as compared to warfarin for the prevention of stroke in AF patients [7-9].

Unfortunately due to increased bleeding risks, some patients cannot tolerate anticoagulation therapy. In patients with non-valvular AF, 91% of left atrial thromboses were isolated to the Left Atrial Appendage (LAA) [10]. Due to this, there has been a great interest in LAA closure devices in AF patients who are poor candidates for oral anticoagulation but are at an increased risk for CVA. LAA closure using the LARIAT suture delivery device (Sentre HEART Inc., Redwood City, California) can be performed on patients with a high risk for CVA with or without contraindications to anticoagulation. Greater interest has been placed on high risk patients for CVA who are not candidates for anticoagulation [4].

We present a case report on a patient who underwent LAA closure using the LARIAT suture delivery device, as she was deemed too high risk for oral anticoagulation. Post procedurally, the patient developed a late pericardial effusion seen by Trans-Thoracic Echocardiogram (TTE). Due to patient's reluctance to undergo pericardiocentesis, she was treated conservatively with steroids with resolution of her pericardial effusion.

## Case

An 81 year old female with a past medical history of frequent falls complicated by Subdural Hematoma (SDH) and concussion, hypothyroidism, and dementia presented with paroxysmal AF with a CHADSVASC of 3 as a referral for evaluation for LAA closure. She was previously treated with warfarin, but in recent years, she was noted to have frequent falls with one resulting in a SDH subsequently leading to her warfarin being discontinued. LAA closure using the epicardial suture delivery device was discussed with the patient and her daughter including the risks, benefits, and alternatives. Both the patient and her daughter expressed interest in proceeding with pre-procedure workup and to undergo the procedure.

Pre-procedure cardiac Computerized Tomography (CT) was obtained, which showed the patient's LAA size and orientation were amenable to closure using the epicardial suture delivery device. Pre-procedure Trans-Esophageal Echocardiogram (TEE) was obtained, which did not show any evidence of LAA thrombus. She subsequently underwent closure of the LAA using a percutaneous subxiphoid approach to deliver a permanent suture around the epicardial base of the LAA as previously well describe [4]. The procedure was successful and the patient tolerated it without difficulty. A post-procedure TTE was obtained, which showed a trivial anterior pericardial effusion (Figure 1A). Output from the pericardial drain was minimal over the next 24 hours and was removed on post-procedure day 1. She was subsequently discharged home with twice daily colchicine and outpatient follow up was arranged.

The patient returned to the Emergency Department (ED) three hours after being discharged from the hospital with shortness of breath with ambulation, light headedness, and sudden sharp substernal chest pain associated with vomiting. In the ED, she was noted to be hypoxic requiring a non-re breather face mask. CT angiography of chest was obtained, which showed extensive bilateral Pulmonary Emboli (PE). Patient was not deemed a candidate for systemic Tissue Plasminogen Activator (tPA) and was placed on systemic intravenous heparin. She underwent emergent successful thrombectomy of the left main pulmonary artery with residual thrombus burden in the sub segmental right pulmonary arteries. An Inferior Vena Cava (IVC) filter was also placed at the end of the case.

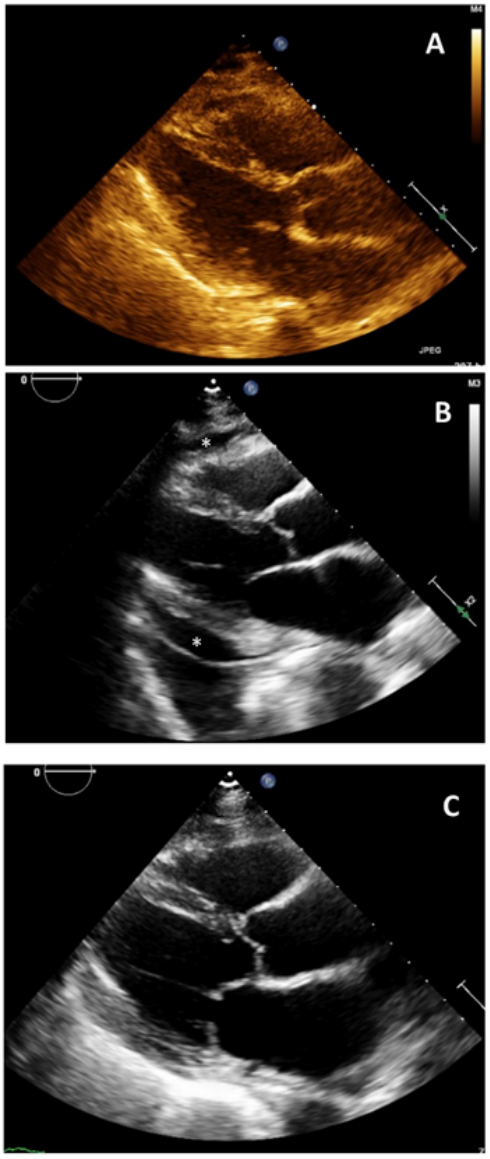


**Figure 1:** Transthoracic Echocardiogram (TTE). Parasternal Long axis view.
(A): Post-procedure Day 1 TTE.
(B): 3 weeks Post-Procedure TTE showing moderate pericardial effusion (effusion shown with asterisks).
(C): TTE after 1 week of steroid initiation showing resolution of pericardial effusion.

Post procedure TTE was obtained, which showed an increase in Right Ventricular (RV) size and reduced function but did not show any pericardial effusion. Patient was subsequently transitioned to apixaban for acute treatment of a pulmonary embolus and was discharged home with Electrophysiology (EP) outpatient follow up. The patient presented to the EP clinic for her post procedure visit complaining of a cough that was exacerbated by lying in the supine position. She was no longer taking colchicine at the time of her clinic visit, although this had been prescribed for a period of six weeks after the procedure. A TTE was obtained, which revealed a moderate circumferential pericardial effusion (Figure 1B). A pericardiocentesis was offered to the patient, but patient refused to undergo any further procedures. She was restarted on colchicine and started on oral lasix with follow up in 1 week.

During her one week follow up, the patient continued to have worsening cough and a repeat TTE showed stable moderate pericardial effusion without tamponade physiology. Pericardiocentesis was again discussed with the patient, but she again declined. A tapering dose of oral methyl prednisolone was then initiated. She was followed up in one week with a repeat TTE, which showed almost complete resolution of her pericardial effusion (Figure 1C). Her symptoms of positional dyspnea, fatigue, and decreased exercise tolerance had resolved as well. Repeat echocardiography three months later demonstrated that the effusion did not recur.

## Discussion

As mentioned above, the incidence of atrial fibrillation is projected to increase over the next decade. With an aging population, a significant number of those patients will have contraindications to anticoagulation for CVA prophylaxis. As a consequence, non-pharmacologic treatment for the prevention of CVA in this population will continue to become more common. Currently, there is great interest in percutaneous, epicardial LAA closure using the LARIAT suture delivery device in patients who are not candidates for anticoagulation.

In patients who undergo this procedure, post-procedure pericardial effusion is relatively common with the incidence being between 10% to 44% [11,12]. In one study, 25% of the pericardial effusions were attributed to LAA perforation and laceration, 25% were attributed to pericardial access (right ventricular perforation or other bleeding during pericardial access), and 50% did not have an attributable cause [11]. The definite therapy for LAA or RV perforation would be drainage of the pericardial space, which is usually not an issue as a pericardial drain is left in place post procedure. Little is known about the 50% of cases with no attributable cause to the effusion.

In our patient, it is unlikely that she had LAA perforation or laceration or RV perforation as her pericardial drain output was minimal. When she represented with a PE, a repeat TTE was obtained approximately 72 hours after her initial procedure and did not show any evidence of a pericardial effusion. The late presentation of the effusion effectively rules out mechanical injury as the etiology of the effusion.

The pericardial effusion that our patient developed post procedure was likely inflammation mediated and similar to that seen in surgical patients with Post Pericardiotomy Syndrome (PPS). Approximately 88% of PPS patients develop pericardial effusions. The usual diagnosis of the pericardial effusion is 3-4 weeks post- procedure [13], which is similar to our patient, who was noted to have pericardial effusion three weeks post-procedure. Our patient was placed on colchicine without improvement in her effusion. Steroids were then initiated, which resolved her effusion as well as her symptoms. In patients with PPS and pericardial effusions, those who are on colchicine and an anti-inflammatory medication (NSAIDs or steroids) have a lower risk of adverse outcomes and a less incidence of further procedures (e.g. pericardiocentesis) [13].

Colchicine has been shown to reduce the incidence of pericardial effusion in PPS patients [14]. Our patient was discharged on colchicine post procedure; however, after being hospitalized for a PE and on her subsequent EP clinic follow up, she was no longer taking colchicine. This may explain why our patient did not have a pericardial effusion on the TTE obtained when she was diagnosed with a PE, but it later developed and was seen on a repeat TTE three weeks after the procedure. In addition, once our patient was started on anticoagulation, it is possible that she had small amount of bleeding in the pericardial space owing to the recent instrumentation of this area. Blood has been shown to be a

potent inducer of inflammation in the pericardial space and this may well have contributed to her pericardial effusion as well.

We propose that the 50% of non-attributable pericardial effusions seen patients undergoing LAA closures using an epicardial, percutaneous suture delivery device may be secondary to pericardial inflammation with similar pathophysiology to PPS. Other hypothesis would be that the ligation of the LAA itself could be the source of pericardial inflammation as this tissue dies and is resorbed over time. At times, physicians have been reticent to use steroids in this patient population owing the concern for an increased risk of bleeding. Our patient was treated similarly to PPS patients with steroids with resolution of her pericardial effusion. In patients who undergo LAA closure with LARIAT device, steroids may have a role in treating pericardial effusions, especially when conservative management is preferred. Further studies need to be done to assess the role of colchicine in preventing post LARIAT procedure pericardial effusions and to assess the role of steroids in non-traumatic (i.e. LAA or RV perforations) post procedure pericardial effusions.

## Author contributions

Dr. Shah and Dr. Maddox have contributed equally to the manuscript. Both authors are thoroughly familiar with the case and the data, have thoroughly read the manuscript, are responsible for the contents, and have approved the manuscript for submission.

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