Radiology Case Reports 19 (2024) 1386–1390

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journal homepage: www.elsevier.com/locate/radcr

Case Report

# Late thrombosis of Gore Cardioform septal occluder device in a patient with history of patent foramen ovale closure ☆

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ARTICLE INFO

*Article history:*
Received 9 December 2023
Accepted 26 December 2023

*Keywords:*
Patent foramen ovale closure
Gore cardioform septal occluder device
Device thrombosis

ABSTRACT

Patent foramen ovale (PFO) is a flaplike opening in the fossa ovalis; between the septum primum and secundum. It is highly prevalent with approximately 25% of the population having a PFO. It is usually asymptomatic but can rarely cause paradoxical embolism leading to stroke and/or significant right to left shunting causing hypoxia. The complications of PFO closure with a percutaneous device include embolization, cardiac perforation, and thrombosis. These are all early complications. We describe the case of a patient with a history of PFO closure who had device thrombosis 3 years after implantation. Management includes anticoagulation with warfarin. Repeat cardiac imaging to document the resolution of thrombosis is recommended.



## Introduction

Device thrombosis is a rare but known late complication of PFO/ASD closure devices, with 1 study estimating an incidence of 0.6% at 6 months. The incidence is higher in the initial period after implantation but decreases with time. Dual antiplatelet therapy is employed for 6 months after initial implantation to prevent such a complication. It is important to note, however, that device thrombosis can occur years after initial implantation, as in our patient.

## Case report

A 76-year-old man with a past medical history of patent foramen ovale (PFO) closed with Gore Cardioform #25 septal occluder device in 2019 and advanced chronic obstructive pulmonary disease (COPD) was admitted to the intensive care unit for acute hypoxic respiratory failure.

His past medical history was significant for PFO closure 3 years prior to presentation. At that time, the patient presented with worsening dyspnea on exertion. A transthoracic

☆ Competing Interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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https://doi.org/10.1016/j.radcr.2023.12.047

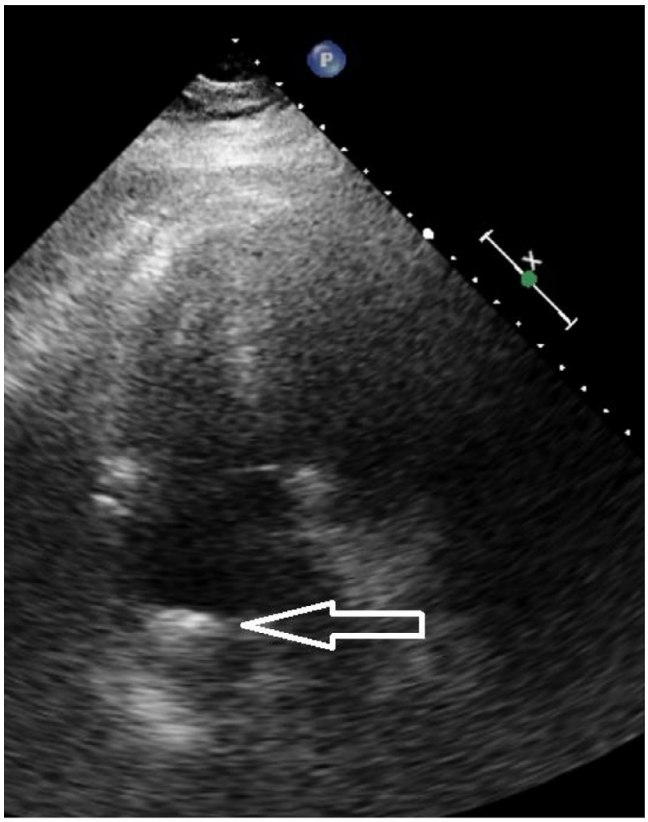

**Fig. 1 – Transthoracic echocardiogram showing an echodensity in right atrium (arrow).**

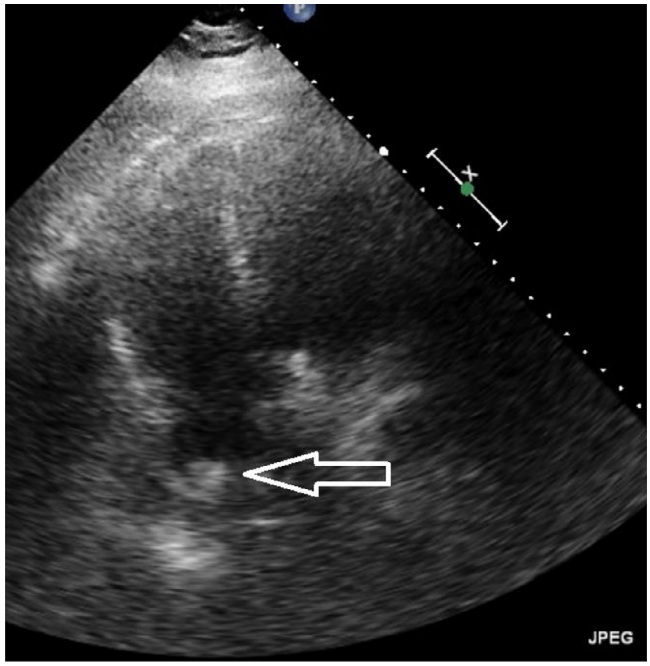


**Fig. 2 – Transthoracic echocardiogram showing an echodensity in right atrium (apical 4 chamber view).**

echocardiogram (TTE) showed a PFO which was confirmed with a transesophageal echocardiogram (TEE). He was found to have secondary polycythemia with a hemoglobin of 19 g/dL. To assess whether the PFO was contributing to his symptoms, an exercise stress echocardiogram was performed which showed worsening hypoxia and increased right to left shunting with reproducibility of symptoms. The patient subsequently underwent PFO closure with Gore Cardioform #25 septal occluder device without peri-operative or postoperative complications. He was prescribed dual antiplatelet therapy (DAPT) with aspirin and clopidogrel for 6 months. A follow-up TTE at 1 and 2-year intervals was normal. His hemoglobin improved to 16 g/dL after PFO closure.

In this admission, the patient's vital signs showed normal blood pressure and tachycardia. He was placed on non-invasive ventilation with continuous positive airway pressure (CPAP) but ultimately required intubation. Labs were remarkable for an elevated D-dimer and a hemoglobin of 15.1 g/dL. Serum troponin was within normal limits. B-type natriuretic peptide was normal. He underwent a computed tomography angiogram (CTA) of the chest which revealed subsegmental pulmonary embolism of the right middle lobe. Lower extremity venous duplex ultrasound did not show any deep or superficial venous thrombosis.

A transthoracic echocardiogram (TTE) was performed which showed preserved ejection fraction and an echodensity in the right atrium (Figs. 1 and 2). To further assess this echodensity, a transesophageal echocardiogram (TEE) was subsequently performed which showed a 3.1 x 3.4 cm mobile mass attached to the PFO closure device in the left atrium (Figs. 3 and 4) but no echodensity was identified in the right atrium on the TEE. This mass was thought to be a thrombus due to its location and attachment to the PFO closure device. It was less likely vegetation due to its morphology and the fact that the patient had negative blood cultures, a normal white blood cell count and did not have a fever. Similarly, suspicion of a tumor was low due to its attachment to the device and morphology. The patient was started on intravenous (IV) heparin and eventually transitioned to oral warfarin to attain an international normalized ratio (INR) of 2.0 to 3.0. Hypercoagulable workup was performed and was negative. His respiratory failure improved with anticoagulation and supportive therapy. A repeat TEE 2 months later showed complete resolution of the thrombus (Figs. 5 and 6). Patient was continued on warfarin.

## Discussion

Complications of percutaneous device closure of atrial septal defects and patent foramen ovale include malposition, embolism, supraventricular arrhythmias, device thrombosis and in some cases, death [1].

Device-related thrombosis is a rare complication of PFO closure, with incidence ranging from 0.2% to 2% [1–3].

In a study of 417 patients that underwent secundum type ASD closure with CardioSEAL/STARFlex and Amplatzer septal occluder devices between 1996 and 2001, 34 patients (8.6%) experienced complications. The most common com-

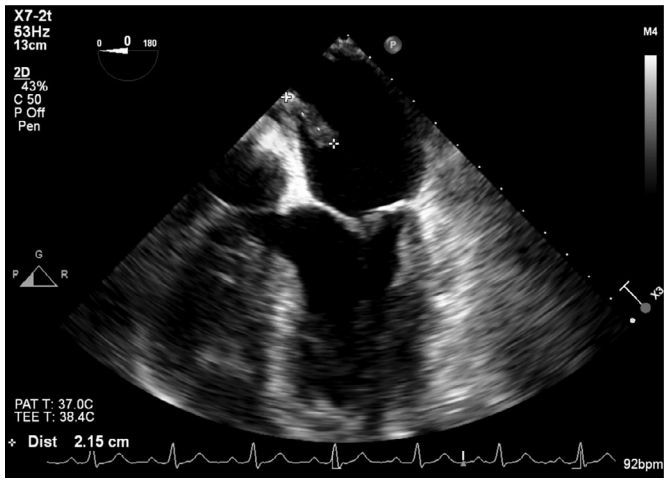


**Fig. 3 – Transesophageal echocardiogram showing an echodensity attached to the Gore cardioform septal occluder device.**

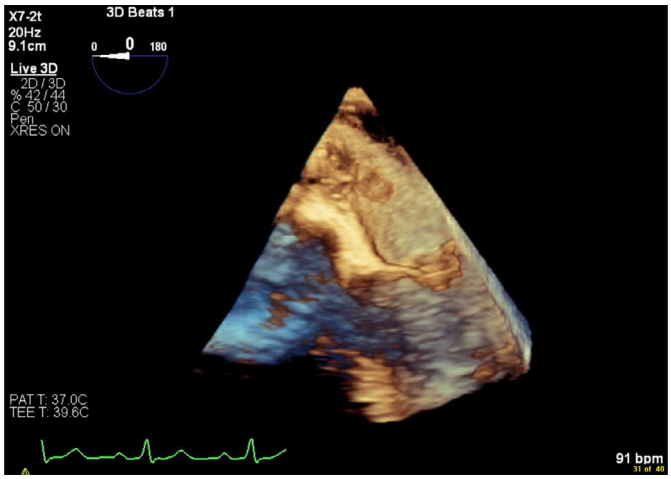


**Fig. 4 – Transesophageal echocardiogram showing an echodensity attached to the Gore cardioform septal occluder device, 3D view.**

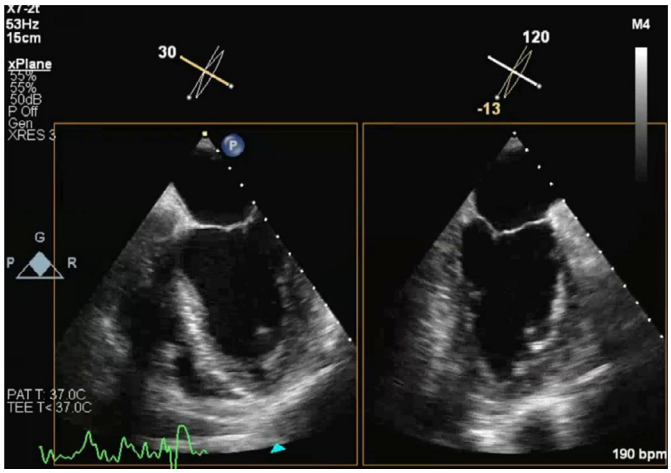


**Fig. 5 – Transesophageal echocardiogram 2 months later showing resolution of thrombus.**

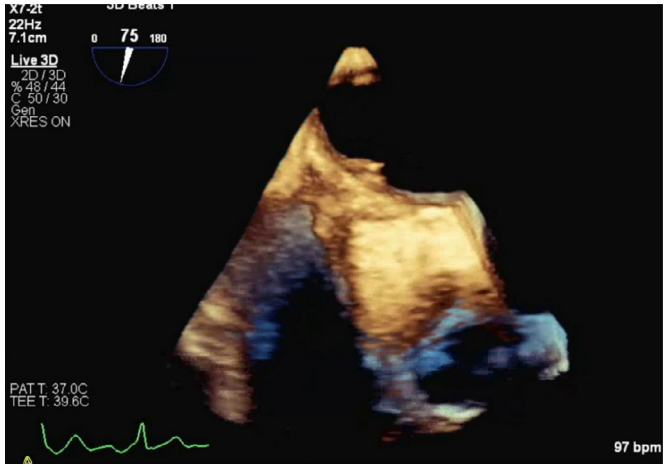


**Fig. 6 – Transesophageal echocardiogram 2 months later showing resolution of thrombus, 3D view.**

plication, about 10 patients, experienced malposition or embolization of the device. There was 1 patient who had thrombus formation on the left atrial disc. The thrombus was seen immediately after delivery of the device despite the patient being anticoagulated during the procedure. The patient received anticoagulation therapy for 6 months with complete resolution on a 6-month transesophageal echocardiogram.

In a 2004 study, Krumsdorf et. al described the incidence and clinical course of thrombus formation in 1000 patients that underwent ASD (n = 406) or PFO (n = 593) closure between 1992 and 2003. The overall incidence of thrombosis was 2% (20/1000). Of the 9 different devices used for closure, the incidence of thrombosis was higher for CardioSEAL (7.1%), StarFLEX (5.7%), PFO-Star (6.6%) devices as compared to Amplatzer device (0%, P = <.05). Most of the thrombi were in the left atrium (n = 11) followed by the right atrium (n = 6) and 3 patients had thrombi in both the left and right atrium. The incidence was higher in PFO patients (15/293, 2.5%) as compared to ASD patients (5/407, 1.2%) but the difference was not clinically significant. Fourteen patients had thrombi identified via TEE at 4 weeks, 6 patients had thrombi after 3 months, 2 patients after 1 year and 1 patient after 5 years. The thrombi ranged from 5 to 30 mm in size.

Postoperative atrial fibrillation and persistent atrial septal aneurysm were factors which were more likely to cause postoperative device thrombosis (P < .05). Interestingly, none of these patients had hypercoagulable disorders. The treatment involved anticoagulation with warfarin with repeat TEE to assess for resolution. A few patients (3/20) required surgery for removal of the thrombus.

A recent case report describes a case of device-related endocarditis and device-related thrombosis days after ASD closure and suggests recent COVID-19 infection-induced hypercoagulability may have played a role [4].

Both aforementioned trials, however, do not have patients with Gore Cardioform occluder device. The Gore REDUCE trial in 2017 described the efficacy of PFO closure for prevention of cryptogenic stroke compared with antiplatelet therapy. In this study, the patients randomized to the PFO closure group underwent closure with either the Gore Cardioform Septal Occluder or the Helex Septal occluder. Six patients (1.4%) had serious device-related complications with only 2 patients (0.5%) having device-related thrombosis [3].

To prevent device thrombosis, patients are advised to take dual antiplatelet therapy (DAPT) with aspirin and clopidogrel for 6 months [5]. Patients who present with device-thrombosis require anticoagulation with repeat TEE to document resolution.

In our patient, no predisposing factor could be identified for his device-related thrombosis, especially almost 3 years after PFO closure. The patient completed 6 months of DAPT, and did not have postprocedure atrial fibrillation or persistent atrial septal aneurysm. A hypercoagulable workup was performed and was negative. The patient did not have any history of malignancy. The patient did not have a transient ischemic attack or stroke. The thrombus was discovered incidentally. One possible explanation may be delayed endothelialization of the device. Animal model studies in the past suggested complete endothelialization of the device occurs in 1-3 months [6]. However, there have been multiple [7–9] case reports describing incomplete or no endothelialization [9] of ASD closure devices beyond the 6 months. These case reports all described the Amplatzer device. It is possible that our patient had delayed or no endothelialization which predisposed him to left atrial thrombus.

## Conclusion

In conclusion, thrombosis of Gore cardioform septal occluder device can occur months and even years after implantation, possibly secondary to lack of endothelialization. Patients may be diagnosed incidentally while undergoing evaluation of other conditions. Treatment is with anticoagulation and follow-up imaging to ensure resolution. This case highlights the unique complication of device thrombosis in a patient with a Gore Cardioform occluder device almost 3 years after PFO closure. Further studies are needed to identify patients who would benefit from a longer duration of antiplatelet therapy or even anticoagulant therapy.

## Patient consent

Written consent was obtained from the patient for the publication of this case report. The patient had the opportunity to read and comment on the manuscript.

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