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CASE REPORT

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Two cases of pericardial tamponade due to nitinol wire fracture of a gore septal occluder

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Abstract

Percutaneous patent foramen ovale (PFO) closure is recommended for secondary prevention of paradoxical embolism through a PFO. In the United States, two Food and Drug Administration-approved PFO closure devices are currently available, and the choice depends on operator preference and PFO anatomy. Although these devices are easy to implant, there are several potential complications. As opposed to the Amplatzer PFO Occluder, there has been no published case of atrial erosion with Gore closure devices. This report describes two cases of pericardial tamponade due to perforation of the atrial wall induced by a wire frame fracture of the Gore Helex and Cardioform devices.

KEYWORDS

ASD/PDA/PFO, CLAS—closure, CONA—congential heart disease, adults, PERI—pericardium, SHDI—structural heart disease intervention

1 | INTRODUCTION

Patent foramen ovale (PFO) is implicated in a wide range of medical conditions, including cryptogenic stroke, migraine with aura, decompression sickness, high-altitude illness, and platypnea-orthodeoxia syndrome.[1] Since percutaneous PFO closure has been shown to be both effective and safe in the short-term, the number of symptomatic PFOs that are annually closed is increasing.[2] W.L. Gore and Associates (Flagstaff, AZ) estimates that more than 40,000 Cardioform Septal Occluders have been implanted as of January 2019, and Abbott Laboratories (Lake Bluff, IL) estimates that over 100,000 Amplatzer PFO Occluders have been used to date. The Cardioform Septal Occluder received FDA approval for PFO closure in 2018[3] whereas the Amplatzer PFO Occluder, available outside the United States since 1997, received FDA approval for PFO closure in 2016.[4] Of note, the Helex Septal Occluder, a predecessor of the Cardioform Septal Occluder, received FDA approval for atrial septal defect closure in 2006 and was used off-label for PFO closure but it has been discontinued by the manufacturer.[5] Despite the availability of PFO closure devices for over two decades, data on long term complications are limited. With the increasing use of PFO closure devices, reporting late complications is important to improve physician awareness and patient safety and to ensure optimal patient and device selection. This report describes two cases of late wire frame fracture, one in a Helex Septal Occluder and another in a Cardioform Septal Occluder, associated with pericardial tamponade.

2 | CASE 1

A 55-year-old woman with a PFO-associated stroke underwent PFO closure with a 30-mm Helex Septal Occluder device in September 2015. The 3-month follow-up transthoracic echocardiogram (TTE) was unremarkable, with appropriate device position and without pericardial effusion. Twenty months later, the patient had abrupt-onset severe chest pain followed by a witnessed collapse. At the emergency room, she was found to be hypotensive, with a systolic blood pressure of 70 mmHg, and tachycardic to 120 beats/minute. TTE demonstrated a large pericardial effusion, which was confirmed by chest computed tomography (CT). Ultrasound-guided pericardiocentesis removed 425 ml of blood and relieved the tamponade physiology. Transesophageal echocardiogram (TEE) demonstrated a shunt from the sinus of Valsalva to the right atrium and an appropriately

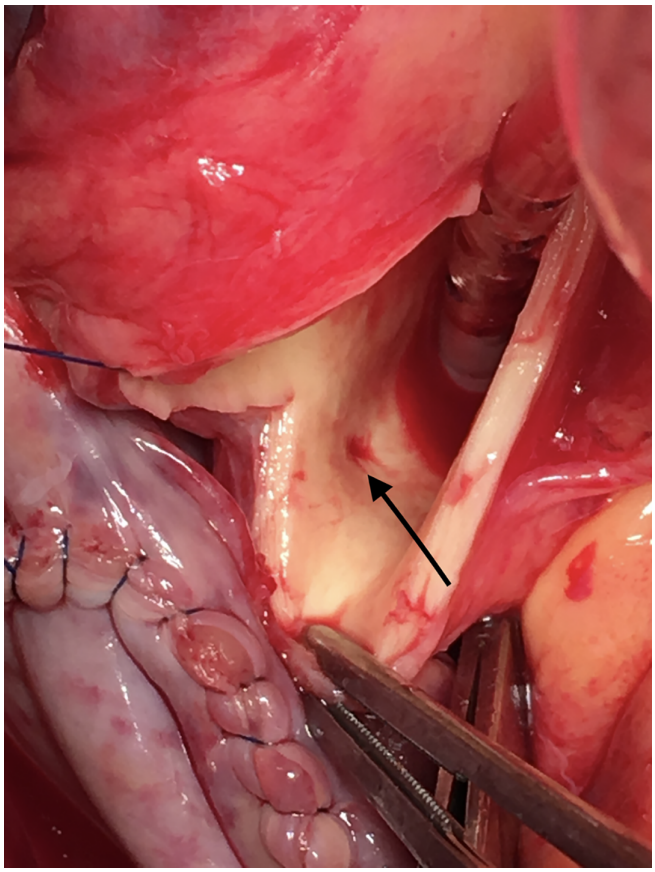

FIGURE 1 Fistula into the Aorta. The aortic root at the commissure between the right and non-coronary cusps had a fistula (black arrow) that extended from the right atrium [Color figure can be viewed at wileyonlinelibrary.com]

positioned septal occluder device without any residual interatrial shunting. Neither the chest CT nor portable anteroposterior (AP) chest x-ray (CXR) showed evidence of device wire fracture.

The occurence of hemopericardium, together with the knowledge that the wire frame of PFO closure devices can fracture, prompted the healthcare team to take the patient to the operating room within a few hours of hospitalization for removal of the PFO closure device through a limited median sternotomy. There was a fistula leading from the right atrium into the aortic root at the commissure between the right and non-coronary cusps (Figure 1). Direct visualization through a right atriotomy revealed a wire fracture. It was assumed that one of the fractured wire ends had penetrated the right atrial medial wall (specifically, the torus aorticus) into the transverse pericardial sinus and then perforated into the aorta (Figure 2). The device was excised (Figure 3) and both the residual atrial septal defect and aortic perforation site were sutured. The recovery course was unremarkable and the patient was discharged on postoperative Day 4.

3 | CASE 2

A 42-year-old woman with a PFO-associated stroke underwent percutaneous PFO closure with a 25-mm Cardioform Septal Occluder device in March 2017. She did well for the next 21 months without chest pain, atrial arrhythmias, or headache. While driving, she suddenly developed severe chest pain, followed quickly by dyspnea and generalized weakness. She was able to drive home where her husband helped her walk into the house. She then lost consciousness and her husband, who described her as blue and pulseless, started cardiopulmonary resuscitation. By the time paramedics arrived, the patient was conscious and had an adequate blood pressure but continued to complain of chest pain. At an outside emergency department, a TTE showed pericardial effusion with tamponade physiology. A pericardial window was performed via a left thoracotomy. The fluid was described as bloody without clots. A workup for potential causes of pericardial effusion, including a chest CT angiogram, was negative for aortic dissection, autoimmune disease, infection, or neoplasm. Three weeks later, she returned to UCLA Medical Center where the Cardioform Septal Occluder device was implanted. TEE showed the Cardioform device to be in the correct position, covered by scar tissue, with a residual effusion in the left pleural cavity, and without evidence of erosion, fractured wire, or residual intracardiac shunting by color Doppler or agitated saline (Figure 4). The CXR also showed the left-sided pleural effusion, which was presumed to be due to continued drainage from the pericardial window (Figure 5). The lateral view CXR showed two fractures in the nitinol wires on the right atrial disc. Cineangiography was performed to better visualize the wire fractures and it confirmed two fractures in the right atrial wires (Figure 6). With the assumption that the fractured wires caused the hemopericardium, the patient was referred to cardiothoracic surgery for removal of the implant followed by patch closure.

Robotic-assisted cardiac surgery was performed through a right thoracotomy. Dense pericardial adhesions were noted. The neointima over the Cardioform Septal Occluder was removed, revealing two wire ends (Figure 7). The surgeon noted that one of the wire ends had been close to the atrium where the perforation likely occurred. A CardioCel patch was used to close the defect. The patient was discharged home on postoperative Day 3. A radiograph obtained during pathologic examination showed that the wire frame of the device was fractured in four different petal locations (Figure 8).

4 | DISCUSSION

In these two cases, based on the visual findings during open-heart surgery, the pericardial effusion occurred secondary to atrial perforation by a fractured wire from the Helex and Cardioform Septal Occluder ~2 years after implantation. While fractured wires in the Helex Septal Occluder (composed of a nickel-titanium alloy) and StarFLEX-CardioSEAL (composed of a cobalt-based alloy) have been described, it had been assumed that these wire fractures were benign because the wires were surrounded by a polymer (expanded polytetrafluoroethylene [ePTFE] in the case of Helex Septal Occluder and polyethylene terephthalate [PET] in the case of StarFLEX-CardioSEAL) and covered by scar tissue.[6,7] Qureshi et al. reported a case of an 8-year-old boy who underwent closure of a secundum atrial septal

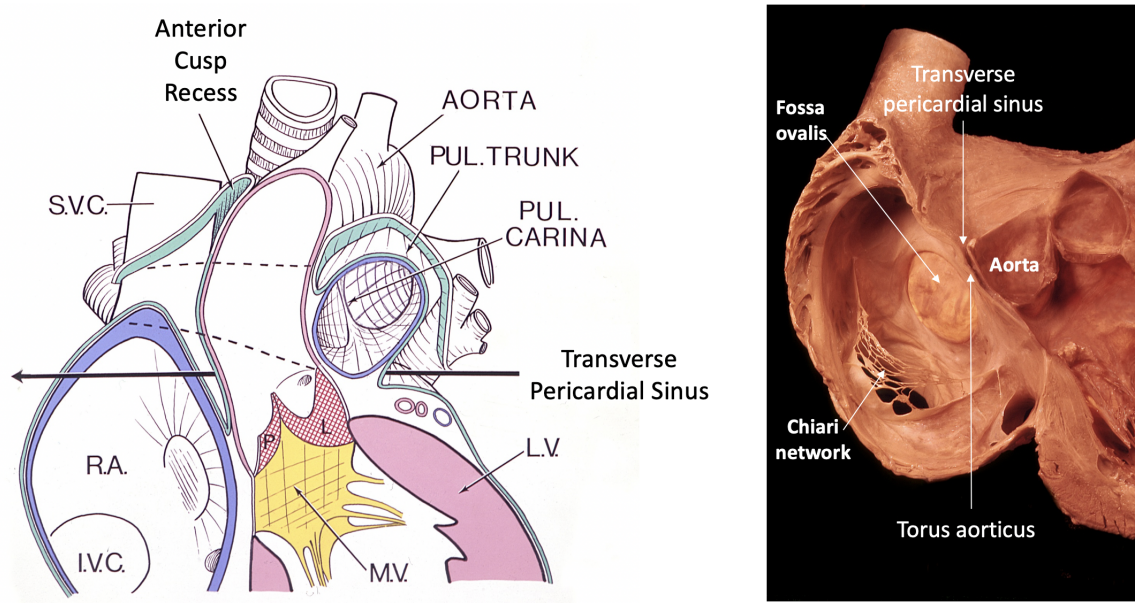

FIGURE 2 The Transverse Pericardial Sinus. (a). A schematic showing that the transverse sinus lies posterior to the ascending aorta and medial to the right atrium. (b). An anatomical depiction of the heart showing that the transverse sinus communicates with the pericardial recess in front of the aorta in the medial aspect of the right atrium. The aortic bulge into the RA is called the "torus aorticus." IVC, inferior vena cava; LV, left ventricle; MV, mitral valve; Pul, pulmonary; RA, right atrium; SVC, superior vena cava [Color figure can be viewed at wileyonlinelibrary.com]

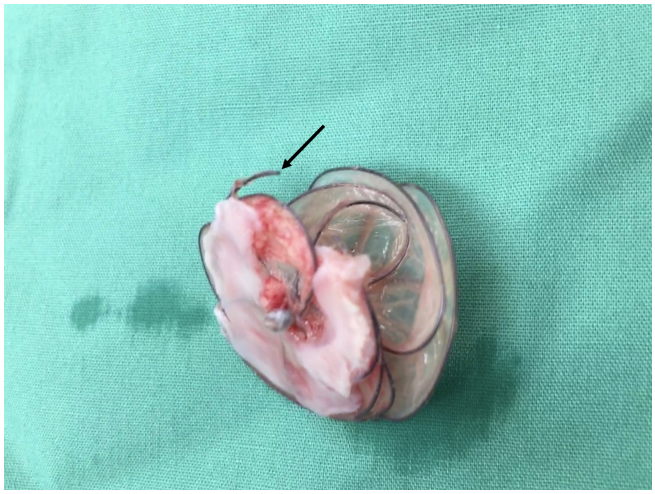

FIGURE 3 Retrieval of the Helex Septal Occluder. A postoperative picture of the PFO closure device shows one wire frame fracture (black arrow). PFO, patent foramen ovale [Color figure can be viewed at wileyonlinelibrary.com]

occluder (ASD) with a Helex device and subsequently had it removed 6 weeks later due to the presence of a wire frame fracture resulting in perforation of the anterior leaflet of the mitral valve.[8] To date, this is the only published report of a wire fracture-induced hemopericardium. This explains why the second case was not immediately referred for surgery or back to the implanting physicians (because of the belief that the hemopericardium could not have been a consequence of the device since it had previously never been reported with

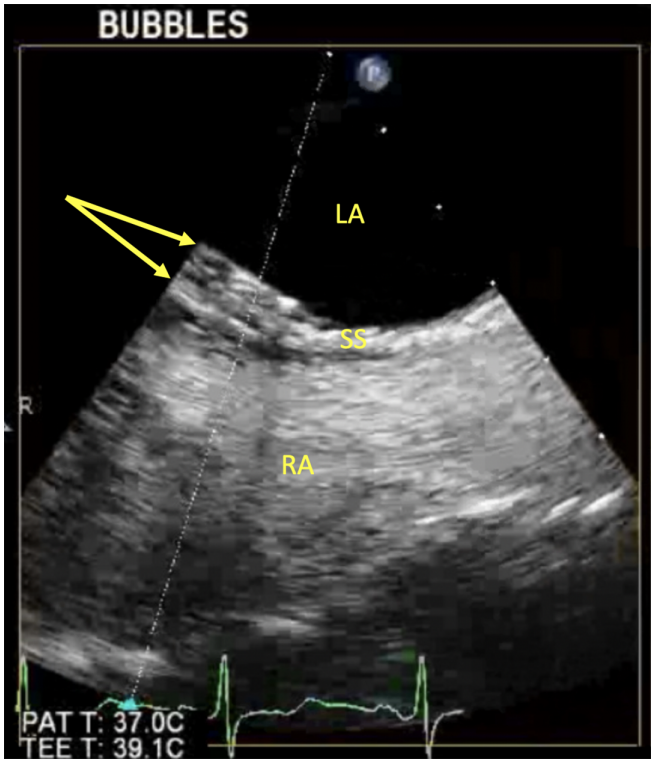

FIGURE 4 Post-PFO closure TEE Bubble Study. TEE showing the RA filled with echoes from an intravenous injection of agitated saline and the Cardioform Septal Occluder (yellow arrows) to be well-seated, without any evidence of residual shunting, and no obvious wire fractures. LA, left atrium; RA, right atrium; SS, septum secundum [Color figure can be viewed at wileyonlinelibrary.com]

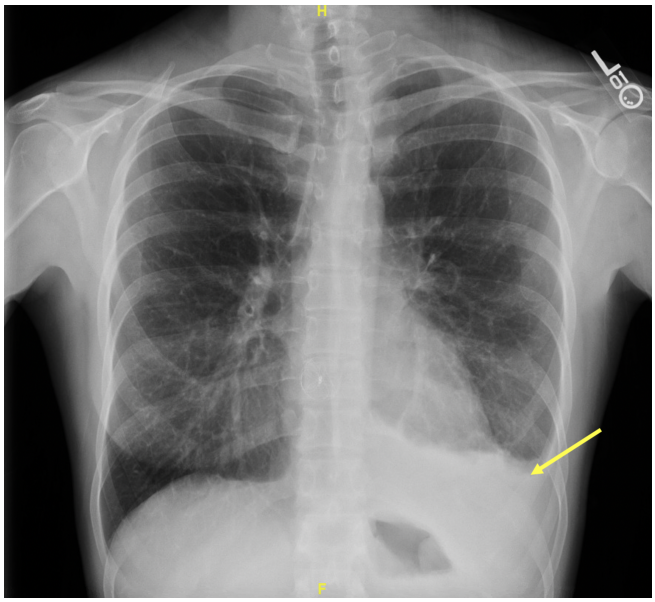

FIGURE 5 Post-PFO closure CXR. CXR showing a moderately-sized left pleural effusion (yellow arrow). CXR, chest radiograph; PFO, patent foramen ovale [Color figure can be viewed at wileyonlinelibrary.com]

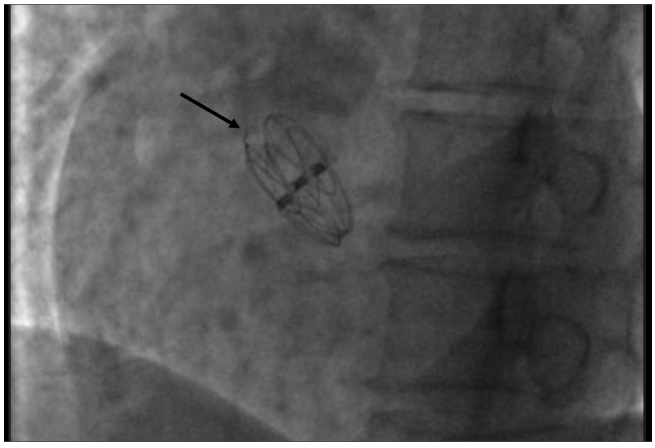

FIGURE 6 Post-PFO closure fluoroscopy. A left anterior oblique cranial view of the Cardioform Septal Occluder with evidence of a superiorly-pointing wire frame fracture on the right atrial disc (black arrow)

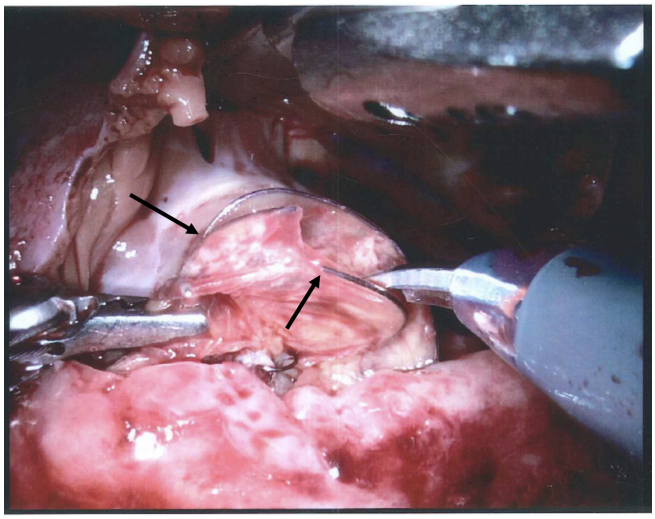

FIGURE 7 The Cardioform Septal Occluder during robotic-assisted surgery. An intraoperative image taken during the robotic-assisted cardiac surgery showing the PFO closure device wire frame to be fractured in at least two different locations (black arrows) [Color figure can be viewed at wileyonlinelibrary.com]

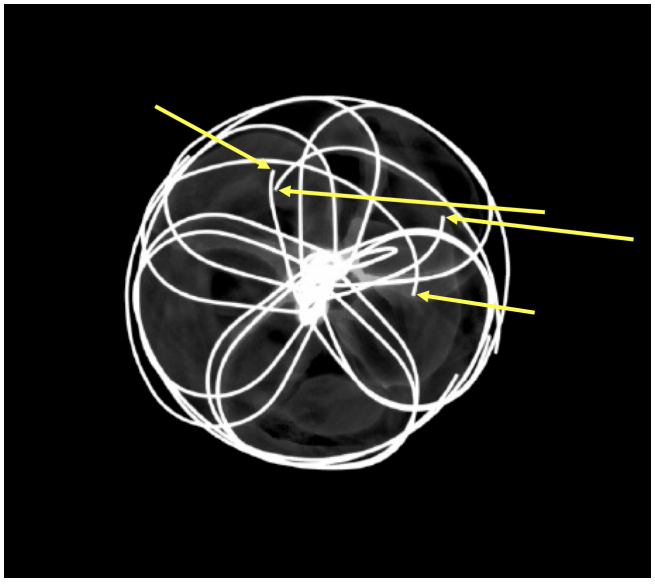

FIGURE 8 Postremoval radiograph of the Cardioform Septal Occluder from Case 2. This radiograph demonstrates four separated wire frame fractures (yellow arrows). There is a total of eight open wire ends [Color figure can be viewed at wileyonlinelibrary.com]

an ePTFE-coated product). There is a higher incidence of wire fractures compared with the incidence of hemopericardium because fractures occurring within the center of the device do not come into direct contact with the cardiac wall. Fractures that occur at the periphery of the device, on the contrary, are adjacent to the cardiac wall and can result in an atrial perforation and subsequent hemopericardium.

Javois et al. reported that at 5-year follow-up of patients who underwent closure of a secundum ASD with the Helex Septal Occluder, wire frame fractures were seen in 14/120 (11.7%) patients.[9] These patients were clinically asymptomatic and 92% of the fractures were detected only by radiographic evaluation involving fluoroscopy. The latter finding is consistent with both cases described in the current report (neither CT nor TEE demonstrated the wire fracture). Furthermore, the AP view CXR in Case 1 was unable to demonstrate a frame fracture whereas the lateral view CXR in Case 2 revealed the fracture, thereby highlighting the importance of obtaining a CXR in the lateral view when trying to identify a frame fracture. Sommer

et al. recently published data on the new Gore Cardioform ASD Occluder for transcatheter closure of ASD. This device is made out of the same material as the Cardioform PFO septal occluder (ePTFE-based polymer over a nitinol wire frame), but it has an anatomically adaptable waist to accommodate defects up to 35 mm in diameter. At 6-month follow-up fluoroscopy, 37/104 (36%) patients were found to have fractured wires, but none developed clinical sequelae within this period.[10] These observations indicate that while nitinol wire fractures are common in septal closure devices, they are thought to be benign.

W.L. Gore and Associates state that the two cases described above are the only cases of hemopericardium reported to the company in ~40,000 Cardioform implants. The only cause of septal closure device-induced hemopericardium described in the literature is erosion from the Amplatzer PFO Occluder device, which is estimated to occur at a frequency of 1 in 20,000 cases.[11]

The FDA stated that the erosion rate of the Amplatzer Atrial Septal Occluder (Amplatzer ASO) is 1–3 in every 1,000 and that an untreated erosion can result in cardiac tamponade, giving some insight into the incidence of pericardial effusion and cardiac tamponade associated with PFO closure devices.[12] The Amplatzer ASO device was the first implantable device to receive FDA approval for closure of ostium secundum atrial septal defects. Amin et al. conducted a review of the registry containing reports of erosion of the Amplatzer Septal Occluder device associated with hemodynamic compromise.[13] Between 1998 and 2004, 23 patients with pericardial tamponade were reported to AGA Medical, the original manufacturer. Although 17/23 (74%) cases occurred within the first 72 hr after the procedure, 5/23 (22%) cases occurred between 5 days and 8 months after the procedure, and 1/23 (4%) case occurred 3 years after the device was placed, highlighting the variability in onset. Most of these patients were treated with surgery for device removal and repair (17/23) but some patients underwent pericardiocentesis (4/23) while a few patients did not have an intervention (2/23). The paper concluded that a deficient aortic or superior rim and use of an ASD device whose diameter is 2 mm greater than the balloon-stretched diameter of the ASD increased the risk of device erosion.

The cause of nitinol wire fractures is believed to be due to metal fatigue from flexing with each heartbeat. Thicker wires might prevent this but would produce a stiffer product that could predispose to device erosion. Fagan et al. reviewed 298 cases of Helex Septal Occluder implants to understand the risk factors associated with wire fractures and the only significant predictor of a wire fracture by multivariate analysis was device size ≥30 mm.[6] Javois et al., Carminati et al., and Sommer et al. also found that the incidence of device fracture correlated with increasing device size.[7,9,10] Of note, the patient in Case 1 received a 30-mm device, the second largest size available for Helex Septal Occluder whereas the patient in Case 2 received a 25-mm device, an intermediate size.

A multicenter international study that reviewed 13,376 percutaneous PFO device implantations found that 38 (1 in 352) devices had to be surgically removed for various reasons, including chest pain, residual shunt, and presence of thrombus on the device.[14] This information, in addition to the possibility of atrial perforation from a metal frame fracture, should be shared with patients considering percutaneous PFO closure.

5 | LIMITATIONS

The following limitations should be acknowledged about this case series. First, the Helex device is no longer manufactured, but it is still of significance for long-term follow up of previous patients. Second, Case 2 did not present to UCLA Medical Center until 3 weeks after experiencing cardiac tamponade, but she had a pericardial window, which likely contributed to her hemodynamic stability during this interim period. Third, Case 2 received chest compressions, which could have caused wire fractures or increased the number of wire fractures but there is no engineering data from W.L. Gore to indicate that this is likely. Although CPR might have aggravated the cardiac tamponade, the lack of radiographic evidence of rib fractures, the most common chest compression-associated injury with an incidence of 25.9% (242/933), makes it less likely that the chest compressions contributed to the presentation.[15]

6 | CONCLUSION

Although percutaneous PFO closure has become an accepted therapeutic procedure for various medical conditions, it is not without risk. Wire fractures in the currently available PFO closure devices occur frequently (36% in the Cardioform ASD device), but the detection of an asymptomatic wire fracture should not cause undue anxiety or act as an indication for removal of the device. Atrial perforation from a fractured wire has not been reported previously, but the current two cases demonstrate that hemopericardium with tamponade is a potential complication of these PFO closure devices. Since these life-threatening complications can occur even years after device implant, there is a need to ensure healthcare professionals are aware of these problems so that they can recognize them promptly, thereby expediting treatment. Symptoms of sudden pericardial effusion and the potential need for surgical removal of a PFO closure device should be explained to all patients during the informed consent process.

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