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Employing Amplatzer Occluder® in Cardiac Free Wall Rupture Repair: A Scoping Study

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Abstract

Cardiac free wall rupture (CFWR) is an uncommon complication of myocardial infarction, cardiac-based procedures, and blunt chest trauma. Cardiac tamponade and shock which occurs as a result of CFWR results in a high mortality rate. Despite the high mortality rate, there is a window of opportunity for intervention in selected patients with acute or subacute free wall rupture. Hence, prompt diagnosis and intervention are key to prevent cardiac tamponade and death. Even though emergency surgical repair is the standard treatment for the CFWR, the catheter-based procedure has provided an alternative treatment option, especially, in the high-risk surgical patients. For instance, Amplatzer occluder® (AO), a device which is used in repairing congenital septal wall defect, is being used as an alternative method of treatment in CFWR. In this systemic review, we assessed the 19 cases of CFWR occurring after invasive cardiac procedures who underwent repair with the utilization of AO®. The study shows that the successful rate of percutaneous closure of CFWR was 84.3% (16/19) with a mortality rate of 15.7% (3/19) in this cohort. Therefore, the in-hospital mortality rate of CFWR closure is comparable with the average in-house mortality rate of emergency surgical repair which is 14%. Furthermore, we found that AO® placement technique has a lower mortality rate compared to the other less-invasive methods such as percutaneous intrapericardial fibrin-glue injection which has a mortality rate of 25%. In conclusion, employing AO® in CFWR repair not only serves as the treatment of choice in the high-risk surgical candidates but could also be applied as an alternative method in the general population. However, further studies are required to assess the outcome and mortality rate of using AO® in CFWR to provide us with a more consistent and accurate data.

Keywords

Amplatzer occluder; Cardiac free wall rupture; acute myocardial complication; catheter-based procedure; surgical repair mortality

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1. Introduction

Cardiac free wall rupture (CFWR) is a rare medical condition which is mostly related to acute myocardial infarction (MI) [1,2]. Even though the prevalence of postinfarction CFWR is only 2–4%, the autopsy studies reveal that it is associated with 7–24% of post MI deaths [3]. In addition to MI, CFWR may occur following cardiac-based procedure and blunt trauma with the prevalence of 0.5–1% and 0.3–1.1%, respectively [2,4].

There are several identified clinical forms of CFWR [5]. The catastrophic form which is the most common one is the sudden rupture of free wall with massive hemorrhage into pericardial space followed by cardiac tamponade and death [5,6]. However, in the subacute form which includes one third of the cases, the course is gradual with slow progressive bleeding into the pericardium [5]. Cardiac pseudoaneurysm, an uncommon form of CFWR, generally is a consequence of myocardium rupture which is contained within a portion of pericardium, therefore the wall of the pseudoaneurysm consists of pericardium and fibrotic tissue [5,7].

Despite the high fatality rate of CFWR, with more than 90% of the patients of developing cardiac tamponade, there is a constrained time period for intervention in patients with acute or subacute wall rupture [8,9]. Hence, prompt diagnosis and proper closure is crucial to prevent cardiac tamponade and death. The availability of point of care echocardiography has resulted in an increased and prompt recognition of cases of CFWR [5]. The standard treatment is emergency surgical repair which is categorized into two different techniques; sutureless repair and sutured repair with the peri-procedural mortality rate of 14% and 13.8%, respectively [8].

Recently, catheter-based procedures have provided alternative treatment options, especially, in high-risk surgical candidates [4]. For instance, transcatheter closure with Amplatzer occluder® is an established method of sealing congenital septal defects [10]. The AO® consists of two self-expandable uniformly sized discs of polyester material (Nitinol wire) that promotes occlusion and tissue growth. Once the area of the rupture wall has been located, the first disc is deployed on the outer aspect of the ruptured cardiac wall; the connecting waist centers the device and allows the second polyester disc to be deployed on the endocardial size of the cardiac wall defect. The AO® is available in a wide range of sizes and can treat defects from 3 mm up 38 mm. Even though the AO related thrombosis is uncommon, the complication might be serious [11]. Therefore, optimal antiplatelet treatment with the proper echocardiographic follow up must be performed, and once the device-related thrombosis is detected, advancing the treatment with therapeutic anticoagulation with aggressive follow-up is inevitable [12]. We performed a systematic review of studies that used AO® in treating CFWR secondary to myocardial infarction, cardiac-based procedure, and trauma in the high-operative risk patients [12].

2. Materials and Methods

A comprehensive computer-based literature search was conducted using PubMed, Google Scholar, CINAHL, Cochrane CENTRAL and Web of Science databases. Our search

keywords “cardiac free wall rupture, ventricular free wall rupture, myocardial infarction, cardiac-based procedure, trauma, Amplatzer Occluder®, transcatheter closure, percutaneous closure” were used to determine cases of CFWR which was treated with percutaneous closure method. This extensive systematic review includes articles from May 2006 until November 2019. The reference list of each article was reviewed and checked for relevant cases.

Only studies using percutaneous closure in treating the cohorts with CFWR were included in this systematic review. We excluded the non-English and non-human studies. In addition, all meta-analysis, review articles, and abstracts were excluded from this study.

Data reviewed included demographic data, cardiovascular risk factor, cause of free wall rupture, location of the perforation, size of Amplatzer, complication of the percutaneous closure and outcome.

3. Results

While 1,810 articles were identified through applying the keywords in database searching, given the title and eligibility criteria, only 24 articles were qualified to be included in this study. After assessing the full-text of the eligible articles, 17 of studies were selected for final analysis.

A total of 19 patients with the median \pm standard deviation (SD) age of 73 ± 16.5 years and a predominance of female gender (52%) were identified (Table 1). The most common cardiovascular risk factors in the study population was coronary artery disease with the prevalence of 68%. The rest of the comorbidities include: hypertension (15%), heart failure (15%), diabetes (5%), and obesity (5%). Invasive cardiac-based procedure was accountable for free wall rupture in 89.4% of the individuals. The other mechanisms involved in CFWR are myocardial infarction and trauma (Table 2). The chamber involved in CFWR was as follows: left ventricle (65%), right ventricle (15%), right atrium (15%), and atrial appendage of left atrium (5%) (Table 1). Amplatzer occluder® was used to fix the free wall rupture in all 19 cases; in 73% (14/19) of patients the AO® percutaneous closure procedure was done without any complication. However, complication during the AO® procedure was reported in 26% (5/19) of cases with the death rate of 60% (3/5) within the cohort who experienced complications. In addition, one of the patients with the successful AO® placement died seven days later due to severe pulmonary contusion and ribs fracture following a traumatic chest injury. Therefore, the successful rate of percutaneous closure of CFWR was 84.3% (16/19) with the mortality rate of was 15.7% (3/19) in this cohort (Table 3).

4. Discussion

To the best of our knowledge, the current systematic review is the first study to assess the mortality rate and complications of employing Amplatzer occluder® in the non-operative management of individuals with CFWR.

CFWR is an uncommon but fatal complication of myocardial infarction with a mortality rate of 39–77% [8,11]. Additionally, CFWR may occur as a complication of cardiac-

based procedure or trauma [4,12]. Significant advancements and increasing use of cardiac-based procedure, such as cardiac catheterization, patent foramen ovale closure, left atrial appendage closure, defibrillator placement, and pericardiocentesis has resulted in an increased incidence of CFWR [4,13–25].

Early detection and prompt intervention is crucial to prevent cardiac tamponade and death [26]. Transthoracic echocardiography plays an important role in detecting pericardial effusion and right ventricular compression which are the most relevant findings in CFWR [8,27]. Detecting echogenic masses in the pericardial spaces significantly enhances both sensitivity and specificity for hemorrhagic effusion. Pericardiocentesis may confirm final diagnosis [8].

Commonly, surgical repair is the definitive treatment for CFWR [28]. There are mainly two different methods of surgery reported in literature; sutureless and sutured repair. The major differentiation between the two groups is employing suture to repair free wall rupture [8]. Matteucci et al [8] reported that the average mortality rate of patients who underwent either type of the surgery was 14%. Recently, less-invasive method, called percutaneous intrapericardial fibrin-glue injection, has been performed in the patients with ventricular free wall rupture with a mortality rate of 25% [29,30]. Terashima et al [31] reported that this less-invasive procedures could apply for the high-risk surgical cases. In accordance with this concept, we performed a systematic review of the cases who underwent AO® placement as a treatment of CFWR that had occurred in its majority as an adverse event of invasive cardiac procedures.

In the current review, the AO® post-procedure complications were reported in 26% of the cases. Pericardial effusion was the most common complication in this cohort with the prevalence of 15.7%, with 2 patients experiencing cardiac tamponade (Table 3). In addition, our analysis revealed that the in-hospital mortality rate of CFWR closure with AO is 15.7% which is comparable with the mortality rate associated with surgical repair, which is the stand of care [8]. Our study findings reaffirm other studies that less invasive percutaneous procedures are a safe and effective alternative in CFWR repair [31]. Additionally, the use of AO® technique has a comparatively lower mortality rate compared to the other less-invasive methods such as percutaneous intrapericardial fibrin-glue injection which report a mortality rate of 25% [8]. Apart from its role in high risk patients, the AO® technique may be considered as a ‘bail-out’ procedure in CFWR. Further evidence is required to investigate its utility in otherwise low risk surgical population

5. Limitations

The major limitation of this systemic review is that it may be subject to selection bias as it is a review focusing on the published literature that report the use of AO® for CFWR that occurred as a complication during an invasive cardiac procedure. Additionally, there are a limited number of cases which used Amplatzer occluder in the treatment of CWFR. All of the studies were either case reports or case series which means there is no control group to compare the procedure more accurately. We only evaluated the in-hospital mortality rate due to lack of information regarding the long-term follow-up of the patients.

6. Conclusion

CFWR carries a high mortality rate. It is an uncommon complication of MI, cardiac procedures, and blunt trauma. The standard treatment of CFWR is emergency surgical repair. The current systematic review demonstrates that less invasive procedures such as using AO in CFWR repair might have the same mortality rate as surgical repair. Therefore, employing AO in the CFWR repair could be a potentially life-saving alternative, especially in patients who are not fit to undergo surgery. However, further studies are required to evaluate the outcome and mortality rate of using AO in the CFWR repair to provide us with more consistent and accurate data.

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Table 1.

Cases of percutaneous closure of cardiac free wall rupture included in this study

	Year, author	Etiology of perforation	Indication of procedure/event that cause free wall perforation	Location of the perforation	Symptoms post perforation
1	2006, Vogel [13]	Cardiac catheterization	HF+EF	LV \bar{c}	Chest pain
2	2006, Gladin [14]	Cardiac catheterization	Enlarging LV pseudoaneurysm	LV	-
3	2007, Harrison [32]	Post MI free wall rupture		LV (posterolateral wall)	-
4	2008, Chiam [15]	PFO closure	Cerebrovascular accident	RAB	-
5	2009, Vignati [4]	CRT	HF+EF	LV	-
6	2009, Alshehri [33]	Cardiac catheterization	LV pseudoaneurysm repair	LV	-
7	2009, Eshtheardi [17]	Cardiac catheterization	VSD closure post LADH MI	LV	-
8	2011, Stolt [18]	Cardiac catheterization	Percutaneous closure of port-a-cath related RA perforation	RA	Retrosternal discomfort
9	2011, Stolt [18]	Cardiac catheterization	Tandem heart placement during cardiogenic shock	RA	-
10	2011, Stolt [18]	Cardiac catheterization	Aortic stenosis	LV	-
11	2012, Acharya [20]	Cardiac catheterization	Percutaneous closure of LV pseudoaneurysm	LV	Chest pain, dyspnea
12	2012, Elbey [19]	Cardiac catheterization	STEMI in inferior and lateral leads	LV (apex)	-
13	2013, Vatan [21]	Cardiac catheterization	Complete occlusion of the right coronary artery	LV (inferior wall)	Chest pain, sweating, blurred vision
14	2014, Meier [22]	Percutaneous LAA closure	LAA closure	LAA	-
15	2015, Tsai [23]	Cardiac catheterization	Percutaneous closure of LV pseudoaneurysm	LV	Dyspnea
16	2016, Saxena [16]	Pericardiocentesis	Cardiac tamponade	RVv	-
17	2018, Dar [12]	Trauma	-	RV (apical)	-
18	2019, Mohammed [24]	Cardiac catheterization	Closure of LV pseudoaneurysm	LV	Chest pain, dyspnea
19	2019, Singleton [25]	RV lead extraction	Recalled RV lead	RV	-

CRT= Cardiac resynchronization therapy; HF+EF= Heart failure with reduced ejection fraction; LAA= Left atrial appendage; HLAD = Left anterior descending artery; ELV = Left ventricle; MI = myocardial infarction; PFO = Patent foramen ovale; RA = right atrium; RV = Right ventricle; VSD = ventricular septal defect; a STEMI= ST elevation myocardial infarction.

Table 2.

Etiology of cardiac free wall rupture

Mechanism	n* (percentage)
Cardiac catheterization	12 (63.1%)
PFO closure	1 (5.2%)
Percutaneous LAA closure	1 (5.2%)
Pericardiocentesis	1 (5.2%)
RV lead extraction	1 (5.2%)
CRT-D placement	1 (5.2%)
Myocardial infarction	1 (5.2%)
Trauma	1 (5.2%)

n = number of patients **PFO**= patent foramen ovale **LAA**=left atrial appendage **RV**=right ventricle **CRT**= cardiac resynchronization therapy defibrillator

Table 3.

Outcome of percutaneous closure of cardiac wall rupture in this study

	Year, author	Size of Amplatzer (mm)	Complications related to Amplatzer occluder placement	Proper intervention after Amplatzer placement complication	Death
1	2006, Vogel [13]	4	-	-	-
2	2006, Gladin [14]	12	-	-	-
3	2007, Harrison [32]	18	-	-	-
4	2008, Chiam [15]	18	-	-	-
5	2009, Vignati [4]	5	-	-	-
6	2009, Alshehri [33]	14	-	-	-
7	2009, Eshthardi [17]	10	-	-	-
8	2011, Stolt [18]	4	Thrombus attached to device nipple	tPAa was given due failure of aspiration of thrombus through the sheath.	
			Five hours later, the patient developed tamponade	Treatment with surgical revision. ASOb was in a stable position without evidence of leakage.	
9	2011, Stolt [18]	5	Tamponade developed 7 days after procedure	Treated with pericardiocentesis.	
			Bacteremia with Staph aureus	Proper antibiotic therapy	
10	2011, Stolt [18]	4	Retropitoneal bleeding	Treatment with surgical revision.	Expired after 2 days from worsening right cardiac failure.
11	2012, Acharya [20]	12	-	-	-
12	2012, Elbey [19]	10,5	Failure of removing the agglutinant pericardial effusion	Treatment with surgical revision	Yes, cardiogenic shock due to hemorrhagic pericardial effusion which was covered in a thick fibrinous peel.
13	2013, Vatan [21]	22	Post procedure long sinus arrest noted.	Atropine injection resolved the sinus arrest	yes, severe hemodynamic deterioration
14	2014, Meier [22]	10	-	-	-
15	2015, Tsai [23]	19	-	-	-
16	2016, Saxena [16]	6	-	-	-
17	2018, Dar [12]	22	-	-	Seven days after due to fatal respiratory failure from significant pulmonary contusions and multiple ribs fracture
18	2019, Mohammed [24]	10	-	-	-
19	2019, Singleton [25]	8	-	-	-

bASO = Atrial septal occluder; a tPA= tissue plasminogen activator