

SYSTEMIC REVIEW ARTICLE

A Comprehensive Systemic Literature Review of Pericardial Decompression Syndrome: Often Unrecognized and Potentially Fatal Syndrome

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Abstract: Background: Pericardial Decompression Syndrome (PDS) is defined as paradoxical hemodynamic deterioration and/or pulmonary edema, commonly associated with ventricular dysfunction. This phenomenon was first described by Vandyke in 1983. PDS is a rare but formidable complication of pericardiocentesis, which, if not managed appropriately, is fatal. PDS, as an entity, has discrete literature; this review is to understand its epidemiology, presentation, and management.

Methodology: Medline, Science Direct and Google Scholar databases were utilized to do a systemic literature search. PRISMA protocol was employed. Abstracts, case reports, case series and clinical studies were identified from 1983 to 2019. A total of 6508 articles were reviewed, out of which, 210 were short-listed, and after removal of duplicates, 49 manuscripts were included in this review. For statistical analysis, patient data was tabulated in SPSS version 20. Cases were divided into two categories surgical and percutaneous groups. t-test was conducted for continuous variable and chi-square test was conducted for categorical data used for analysis.

Results: A total of 42 full-length case reports, 2 poster abstracts, 3 case series of 2 patients, 1 case series of 4 patients and 1 case series of 5 patients were included in the study. A total of 59 cases were included in this manuscript. Our data had 45.8% (n=27) males and 54.2% (n=32) females. The mean age of patients was 48.04 ± 17 years. Pericardiocentesis was performed in 52.5% (n=31) cases, and pericardiostomy was performed in 45.8% (n=27). The most common identifiable cause of pericardial effusion was found to be malignancy in 35.6% (n=21). Twenty-three 23 cases reported pre-procedural ejection fraction, which ranged from 20%-75% with a mean of 55.8 ± 14.6%, while 26 cases reported post-procedural ejection fraction which ranged from 10%-65% with a mean of 30% ± 15.1%. Data was further divided into two categories, namely, pericardiocentesis and pericardiostomy. The outcome as death was significant in the pericardiostomy arm with a p-value of < 0.00. The use of inotropic agents for the treatment of PDS was more common in needle pericardiocentesis with a p-value of 0.04. Lastly, the computed recovery time did not yield any significance with a p-value of 0.275.

Conclusion: Pericardial decompression syndrome is a rare condition with high mortality. Operators performing pericardial drainage should be aware of this complication following drainage of cardiac tamponade, since early recognition and expeditious supportive care are the only therapeutic modalities available for adequate management of this complication.

Keywords: Pericardial decompression syndrome, pulmonary edema, ventricular dysfunction, pericardiocentesis, pericardial drainage, cardiac tamponade.

1. INTRODUCTION

Cardiac tamponade (CT) is a life-threatening condition that occurs secondary to the accumulation of fluid in the

pericardial sac; this causes compression of the myocardium, disrupting the normal cardiac function. Cardiac tamponade presents with shortness of breath, tachypnea, tachycardia, and hypotension. It requires emergent treatment, which is accomplished by two main strategies—namely needle pericardiocentesis and/or surgical pericardiostomy. Both the procedures are employed indistinctly, with the goal of drain-

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ing fluid and improving cardiac hemodynamics. Drainage of pericardial effusion is also indicated for diagnostic purposes and symptomatic relief in the absence of tamponade [1].

Hemodynamic stabilization is the expected outcome after either percutaneous or surgical pericardiocentesis, but in some instances, patients experience paradoxical hemodynamic instability. This phenomenon was first described in 1983 [2]. Multiple terminologies have been designated to this complication, including Paradoxical Hemodynamic Instability (PHI) [3, 4] and postoperative low cardiac output syndrome (POLCOS) [5]. In 2010, Angouras *et al.*, coined the term “Pericardial Decompression Syndrome” (PDS) to address the publication bias for this complication [6].

PDS is a rare but fatal complication of pericardiocentesis [5, 7]. Although PDS as an entity has been reviewed previously [8], the term PDS is scarce and not well-documented in the literature, and PDS requires an update to better understand its epidemiology, presentation, management, and prevention.

2. METHODS

2.1. Literature Search

MEDLINE, Science Direct, and Google Scholar databases were utilized to do a systemic literature search. PRISMA protocol was employed [9]. Abstracts, case reports, case series, and clinical studies were identified from 1983 until April 2019. The MeSH terms that were searched for were pericardial decompression syndrome, cardiac tamponade, paradoxical hemodynamic instability, ventricular dysfunction, pericardial effusion, pericardial window, pericardiocentesis, cardiogenic shock, and pulmonary edema. Inclusion criteria for the literature were as follows: 1) cases and/or case studies discussing patients with pericardial effusion who had successful pericardiocentesis or pericardiostomy and unexpectedly developed hemodynamic instability, 2) cases and/or case studies discussing patients who did not have procedural complications which would otherwise explain hemodynamic instability. In compliance with PRISMA protocol, references of the shortlisted literature were cross-checked in order to avoid publication bias, which previously existed for this topic. The above-mentioned databases were searched by the authors and duplicates were removed.

2.2. Data Extraction

Clinical variables of interest were extracted from case reports and case series. These variables were age, sex, blood pressure prior to the procedure, heart rate prior to the procedure, history of malignancy, presence of pulsus paradoxus, elevation of JVD, type of procedure, total number of procedures, total volume removed, the onset of symptoms, elevation of troponin, use of inotropes for stabilization, outcome of the patient, time of death in relation to the procedure, the result of coronary angiography if performed, pre-procedure and post-procedure echocardiography, type of cardiac dysfunction (LV, RV, or biventricular), pulmonary edema with cardiogenic shock, and pulmonary edema without cardiogenic shock.

2.3. Statistical Analysis

Patient data was tabulated in SPSS version 20. Cases were divided into two categories- surgical and percutaneous groups. The two tests used for statistical analysis were the T-test, completed for the continuous variable, and the chi-square test or fisher's exact test, completed for categorical data.

3. RESULTS

A total of 44 full-length case reports, 2 poster abstracts, 4 case series of 2 patients, 1 case series of 4 patients, and 1 case series of 5 patients were included, totaling 52 cases in this manuscript. The sample was composed of 43.5% (n=28) males and 54.2% (n=34) females; with females having higher predisposition towards mortality (p-value 0.025) (Table 1). The mean age of patients was 47.52 ± 18.04 years. The identifiable causes of pericardial effusion include malignancy (38.7%, n=22), inflammation (14.5%, n=9), tuberculosis (8.1%, n=5), infection in (4.8%, n=3) while the remaining 37.1% (n=23) of cases had no reported etiology. Malignant pericardial effusion had no association with mortality (p-value 0.428). Of the 62 cases, 88.7% (n=55) had cardiac tamponade and 4.8% (n=3) had pericardiocentesis without cardiac tamponade; a case series of 4 patients did not provide sufficient information for 3 patients to be placed in either category [10]. On presentation or prior to the procedure, 29 of 94.12% (n=32/34) of patients had tachycardia. Pulsus paradoxus was present in 32.3% (n=20) of patients, negative for 8.1% (n=5) of the patients, and not specified in 59.7% (n=37) of the patients. Jugular venous distention was present in 46.8% (n=29) of the patients, negative in 3.2% (n=2) of patients, and not specified in 50.0% (n=31) of patients (Table 2).

Twenty-four cases reported pre-procedural ejection fraction, which ranged from 20%-75% with a mean of $56.2 \pm 14.4\%$, while 29 cases reported post-procedural ejection fraction, which ranged from 10%-65% with a mean of $29.6\% \pm 14.5\%$ (Table 3; Table 4). The size of the effusion was large in 77.4% (n=48) of the patients, 4.8% (n=3) reported the size of the effusion as moderate, and 17.7% (n=11) did not comment on the size of the effusion.

Pericardiocentesis was performed in 53.2% (n=33) of the cases, and pericardiostomy was performed in 45.2% (n=28) of the cases (Table 5). One case reported the development of PDS after both procedures on the same patient [2]. Forty-eight cases reported the volume of the pericardial effusion as ranging from 100mL-2760mL with a mean volume of 958.3 ± 524 mls. The presentation of PDS was as follows: 29% (n=18) of the patients had left ventricular failure (LVF), 35.5% (n=22) of the patients had a biventricular failure (BVF), and 6.5% (n=4) of patients had a right ventricular failure (RVF). Eight of the LVF patients had pulmonary edema [4, 11-16], 17 of the BVF patients had pulmonary edema [8, 17-32], 2 of the RVF patients had pulmonary edema [33, 34], 3 patients had non-cardiogenic pulmonary edema [2, 35, 36] and 3 patients had pulmonary edema but there was no post pericardiocentesis echo to specify the origin of the pulmonary edema [37-39]. Troponin levels were tested in 15 patients, out of whom 60% (n=9) were found to have elevated troponin. Post-procedure, 64.5% (n=40) of patients

Table 1. Association of mortality with patient characteristics.

-	Non-Fatal	Fatal	p Value
Age	48.06 ± 17.46 (n=46)	45.71 ± 20.43 (n=14)	0.424
Female Sex (n =34)	22	12	0.025*
Malignancy (n=21)	15	6	0.518

Table 2. Summary of literature review of published case reports and case series.

No.	Author	Year	Pt#	Age	Sex	Bp	HR	PP	JVD	CT	Size	S/P	Vol	Time	Failure	Inotrope	Outcome
1	Vandyke [2]	1983	1	42	M	80/50		+	+	+	L	B	680	Imm		-ve	Survived
2	Shenoy [38]	1984	2	57	M	90/60		+	+	+	L	S	1000	Imm		-ve	Survived
3	Glasser [35]	1988	3	33	M	100/60		+	+	+	L	P	2100	Imm		-ve	Survived
4	Downey [39]	1991	4	50	M		104	+	+	+	L	P	1950	4		-ve	Survived
5	Hamaya [59]	1993	5	16	F	110/70	120			+	L	P	700	Imm		+ve	Survived
6	Wolfe [15]	1993	6	22	F	90/50	140	+		+	L	P	650	12	LV	-ve	Survived
			7	50	F	100 SBP		+		+	L	P	650	Imm	LV	-ve	Survived
7	Braverman [46]	1994	8	27	F	90/60	130		+	+	L	S	1000	Imm	Bi	+ve	Survived
8	Uemura [16]	1995	9	18	M	87/59	120	N/A	+	+	L	P	450	Imm	LV	+ve	Survived
9	Neelakandan [61]	1996	10		F	-	-	-	-	+	L	S	-	6	-	+ve	Death
	-	-	11		M	-	-	-	-	+	-	P	-	-	-	+ve	Survived
10	Anguera [26]	1997	12	68	F	60/40	100	+	+	+	L	P	800	Imm	Bi	+ve	Survived
11	Dosios [10]	1997	13	36	F	-	-	-	-	-	-	S	-	-	-	+ve	Death
	-	-	14	65	F	-	-	-	-	-	-	S	-	-	-	+ve	Death
	-	-	15	31	F	-	-	-	-	+	-	S	-	-	-	+ve	Death
	-	-	16	60	M	-	-	-	-	-	-	S	-	-	-	+ve	Survived
12	Thrush [25]	1998	17	58	M	125/90	145	+	+	+	L	S	600	.25	Bi	+ve	Survived
13	Sunday [17]	1999	18	60	F	110/74	110	+		+	L	S	700	Imm	Bi	+ve	Death
14	Chamoun [56]	2003	19	36	F	-	-	-	-	-	M	P	1070	24	LV	+ve	Survived
	-	-	20	46	F	80/50	110	+	+	+	L	P	1000	24	LV	-ve	Survived
15	Dosios [5]	2003	21	37	F	-	-	-	-	+	-	S	-	-	-	-	Death
	-	-	22	69	F	-	-	-	-	+	-	S	-	-	-	-	Survived
	-	-	23	67	F	-	-	-	-	+	-	S	-	-	-	-	Death
	-	-	24	31	M	-	-	-	-	+	-	S	-	-	-	-	Death
	-	-	25	70	M	-	-	-	-	+	-	S	-	-	-	-	Death
16	Geffory [37]	2004	26	53	M	140/90	110	+	+	+	L	S	1500	Imm	RV	+ve	Death
17	Liou [27]	2005	27	22	F	54/30	-	-	+	+	L	S	500	Imm	Bi	+ve	Death
18	Ligero [28]	2006	28	41	F	-	100	+	+	+	L	P	1000	3	Bi	-	Survived
19	Bernal [11]	2007	29	45	F	-		+	+	+	M	P	500	6	LV	+ve	Survived

(Table 2) contd...

No.	Author	Year	Pt#	Age	Sex	Bp	HR	PP	JVD	CT	Size	S/P	Vol	Time	Failure	Inotrope	Outcome
20	Dosios [31]	2007	30	66	F	80/50	130	-	+	+	L	S	500	12	Bi	+ve	Survived
21	Sharaf [48]	2008	31	55	F	110/75	120	-	+	+	L	P	600		RV	+ve	Survived
22	Sevimil [52]	2008	32	42	F	90/60	110	+	-	+	L	P	500	24	LV	-ve	Survived
23	Ischaki [55]	2008	33	25	M	85/60	110	-	-	+	L	P	1800	3	Bi	+ve	Survived
24	Sunderji [47]	2009	34	56	M			-	-	+	L	P	1500	24	Bi	-ve	Survived
25	Flores [12]	2009	35	80	M	90/40	120	+	+	+	L	P	1200	48	LV	+ve	Survived
26	Karamchalis [36]	2009	36	19	F	120/80	110	-	+	+	L	S	1600	Imm	-	-	Death
27	Lim [30]	2011	37	38	F	130/80		-	-	+	L	S	1000	9	Bi	+ve	Death
28	Lango [23]	2011	38	16	M	125/60	125	-	-	+	L	S	1800	5	Bi	+ve	Survived
29	Al Banna [63]	2011	39	17	M	99/63	114	+	+	+	L	P	450	4	BI	+ve	Survived
30	Abdelsalam [4]	2012	40	65	M	106/75	116	+	+	+	L	S		Imm	LV	+ve	Survived
31	Weijer [53]	2013	41	69	F	120/70	120		+	+	L	P	800	6	LV	+ve	Survived
32	Philippakis [64]	2013	42	62	F	120/80	110	+	+	+	L	S		24	LV	+ve	Survived
33	Pardhan [8]	2014	43	41	M	111/66	115	-		+	L	P	1550	0.5	Bi	+ve	Survived
34	Ayoub [40]	2015	44	62	M	130/90	101	+	+	+	L	P	1800	9	Bi	+ve	Survived
35	Sng [66]	2015	45	65	M	120/60	-	-	-	+	L	P	700	8	LV	+ve	Survived
36	Koener [18]	2015	46	37	F	-	-	-	-	+	L	S	1500	11	BI	+ve	Survived
37	Basamji [20]	2015	47	56	F	100/76	110	-	+	+	L	P	700	Imm	Bi	+ve	Survived
38	Liao [20]	2015	48	54	M	110/96	120	-	-	+	L	S	1200	Imm	Bi	+ve	Survived
39	Versaci [49]	2015	49	78	F	95/70	95	+	-	+	L	P	1000	24	LV	+ve	Survived
40	Takeuchi [14]	2016	50	42	M	131/104	113			+	L	P	700	0	LV	+ve	Survived
41	Fozing [13]	2016	51	44	M	-	-	-	-	+	L	S	2760	3	LV	-ve	Survived
42	Kuroda [57]	2016	52	82	M	106/44	76		-	-	M	P	430	12	RV	+ve	Survived
43	Albeyoglu [21]	2016	53	43	F	110/76	110		+	+	L	S	1000	12	Bi	+ve	Survived
44	Methachittiphan [57]	2016	54	30	M	-	-	-	-	+	L	P	560	12	LV	-	Survived
45	Han [37]	2016	55	84	F	-	-	-	-	+	L	S	600	Imm	Bi	+ve	Death
46	Said A [37]	2017	56	54	M	75/96	-	-	-	+	L	S	794	-	-	-ve	Survived
47	Moon [41]	2017	57	52	F	100/60	119	-	+	+	L	P	900	24	Bi	-ve	Survived
48	Guler [22]	2017	58	27	F	100/60	106	-	+	+		P	980	2	Bi	+ve	Death
49	Klimis [24]	2017	59	33	M	123/83	130	-	+	+	L	P	2000	Imm	Bi	+ve	Survived
50	Chung [34]	2018	60	41	F	-	-	+	+	+	L	S	250	Imm	RV	+ve	Survived
51	Moudgil [65]	2018	61	69	M	-	-	-	-	-	L	P	620	-	LV	-ve	Survived
52	Mahajan [62]	2019	62	37	F	80/60	140	-	+	+	L	P	900	24	LV	-ve	Survived

Legends: M- Male, F- Female, + positive finding, - negative finding, Imm- immediately during the procedure, LV- left ventricular, RV- right ventricular, Bi-Biventricular, S- surgical pericardectomy, P- needle pericardiocentesis, M- medium volume, L large volume.

Table 3. Echocardiographic findings before drainage.

Echo Finding	Pre-Procedural Echo		
	RA	RV	LV
Normal	0	3	23
Collapsed	26	27	3
Dilated	1	2	0
Not mentioned	35	30	34

Table 4. Echocardiographic findings after the drainage procedure.

Echo Finding	Post Procedural Echo		
	RA	RV	LV
Normal	9	4	9
Collapsed	0	1	1
Dilated	3	11	5
Hypokinetic	1	20	28
Not Mentioned	48	33	21

Table 5. A comparison between pericardiocentesis and pericardiostomy.

	Pericardiocentesis	Pericardiostomy	p value
Total Sample	32	28	-
Death	1 (3.1%)	14 (48.3%)	< 0.000
Mean Age	45.53 ± 19.3 (n=32)	50.07± 16.7 (n=27)	0.58
Total Volume	948.8 ± 500.9 (n=32)	1083.6 ± 588.3 (n=15)	0.818
Inotrope Support	65.6% (n=21/33)	86.4% (n=19/22)	0.120
Recovery Time	8.35 ± 4.22 (n=20)	10.71± 6.32 (n= 7)	0.124

required inotropic support for stabilization. Eight patients had cardiac catheterizations performed and all were negative for any significant coronary artery stenosis. Two cases reported ballooning of the ventricles post-procedure [40, 41].

Comparing pericardiocentesis to pericardiostomy; overall mortality was significantly higher in the pericardiostomy group compared to the pericardiocentesis group (p-value <0.0000) (Table 5). Lastly, recovery time was higher in the pericardiostomy group compared with the pericardiocentesis group; however, the higher recovery time in the pericardiostomy group was not statistically significant (p-value 0.275).

4. DISCUSSION

4.1. Risk Factors

Predictors of pericardial decompression syndrome were reported in the past. Our data indicates malignancy-related effusions to be the most common cause, accounting for

38.7% of the cases followed by unidentifiable etiologies in 37.1% of the cases (Table 6). These findings are in concurrence with previous studies done on this subject [7, 42, 43]. Sabzi *et al.*, also reported in their study that the history of malignancy and radiotherapy were the only independent predictors of PDS [7]. It would be reasonable to predict that patients with malignancy have a high likelihood of developing pericardial effusion and not necessarily PDS.

Patient characteristics which increase the likelihood of PDS include female sex, as ascertained by Sabzi *et al.*, in his study (p <0.001). Our data also contains a higher percentage of females (54.7% of cases). The age of the patients ranged from 16 to 84 years of age, with a mean of 47.52 ± 18.04. Procedural characteristics, which predispose the patient to develop PDS, include the amount of volume drained. Wagner *et al.*, reported in their study that patients with mean drainage of 647± 217 ml developed PHI (p-value 0.003) [44]. We found only one case of PDS that resulted from draining pericardial fluids of less than 100 ml [35]. Another

Table 6. Causes for the pericardial effusion.

Cause	Frequency	Percentage
Carcinoma	21	35.6%
Tuberculosis	4	6.8%
Infection	3	5.1%
Inflammation	8	13.6%
Unspecified	23	38.9%

contributing factor might be the rate of drainage of effusion. Most reports recommend slow drainage of effusion. Lastly, the type of procedure is also considered to be an important factor that contributes to the development of PDS. Horr *et al.*, reported hemodynamic instability as an outcome to be 3% (n= 22) in the pericardiocentesis arm and 5% (n=27) in the pericardiostomy arm, with statistical significance of p-value 0.036 [45].

4.2. Clinical Presentation

PDS is a heterogeneous entity; common clinical presentations include sudden hypoxia and pulmonary edema in 80.4% (n=33/41) of cases and/or hypotension requiring inotropes support in 66.7% (n=40/60) of cases. Time of Onset of PDS varies, ranging from immediately after the procedure to 48 hours later, this is in agreement with a previous literature review [8].

Correlation of clinical presentation and echocardiographic findings in the setting of suspected PDS is imperative as biventricular failure was the most common finding in the cases reviewed for this study.

4.3. Pathogenesis

Physiologically, cardiac tamponade results primarily from increased intrapericardial pressure, which leads to compression of all cardiac chambers causing decreased preload and eventually decreased stroke volume and hypotension. The early compensation for this occurs via augmentation of adrenergic tone, causing tachycardia and increased inotropy, thereby improving cardiac output [2, 16, 46]. As the cardiac tamponade worsens and there is consistently reduced stroke volume and hypotension, an increase in systemic vascular resistance occurs [16].

The pathophysiological mechanism that dictates PDS is not completely understood. Numerous hypotheses have been proposed [47]. Pericardiocentesis causes the RV volume to increase significantly at the expense of LV due to the sudden and large increase in venous return with subsequent bowing of the inter-ventricular septal to the LV side, which may lead to acute left-sided heart failure and pulmonary edema [48-50]. Heart failure could be further precipitated as systemic vascular resistance (SVR) may remain persistently high in some cases even after pericardiocentesis and reversal of cardiac tamponade [8].

Another theory suggests that Pericardiocentesis leads to a significant increase in RV volume in comparison to LV; with

no changes in the ejection fraction of the two ventricles, thereby causing LV to overload [51-53]. This is a direct result of the higher right ventricular transmural compliance compared to that of the left ventricle. In addition, the decompression of the effusion would cause a greater increase in the right ventricular strain in comparison to the left. Thus, in the principle of the Frank-Starling mechanism, the right ventricular stroke volume would be greater than the left [54]. Additionally, LV in the setting of acute overload with high filling pressure undergoes “wall stress” (Laplace’s Law) [55]. This is augmented by negative pressure in the pericardial cavity, causing further decompensation and cardiac stunning [55-57]. This mechanism explains the echocardiographic findings of RV dilation and the radiologic finding of pulmonary edema in some in patients. Similar findings were reported by other authors, with evidence from radionuclide and echocardiographic imaging [26, 58].

Our analysis reveals that 53.2% (n=33/62) of the patients had pulmonary edema while 43.5% (n=27) of patients had pulmonary edema alongside ventricular dysfunction. It appears that at least in some patients, pulmonary edema does not occur or occurs due to a different mechanism. Glasser *et al.*, measured pulmonary capillary occlusion pressures immediately after pericardiocentesis when the patient developed pulmonary edema. They reported that the measured pulmonary occlusion pressure was never high enough to support volume overload but postulated that capillary permeability was a more plausible reason for the development of pulmonary edema [35].

Another plausible hypothesis is transient ventricular failure due to ischemia and myocardial stunning. Multiple theories describe the mechanism. First, elevated intrapericardial pressures decrease the coronary blood flow; thus, rapid drainage and drop of surrounding pressures would quickly increase coronary myocardial perfusion, exposing the myocardium to reperfusion injury [59], especially if there is pre-existing coronary artery disease. The second theory suggests the epicardial coronary arteries are compressed, leading to decreased blood flow to the myocardium, which causes ventricular dysfunction [46, 51, 60]. Although these changes occur during the cardiac tamponade, they are not evident until decompression. Wolfe and Edelman suggested that the sympathetic overdrive that occurs during the tamponade masks the ventricular dysfunction by increasing inotropy and tachycardia. They also elaborate that once decompression occurs, sympathetic overdrive decreases, thereby unmasking the ventricular dysfunction [15]. Our analysis reveals that transient ventricular failure is potentially due to myocardial

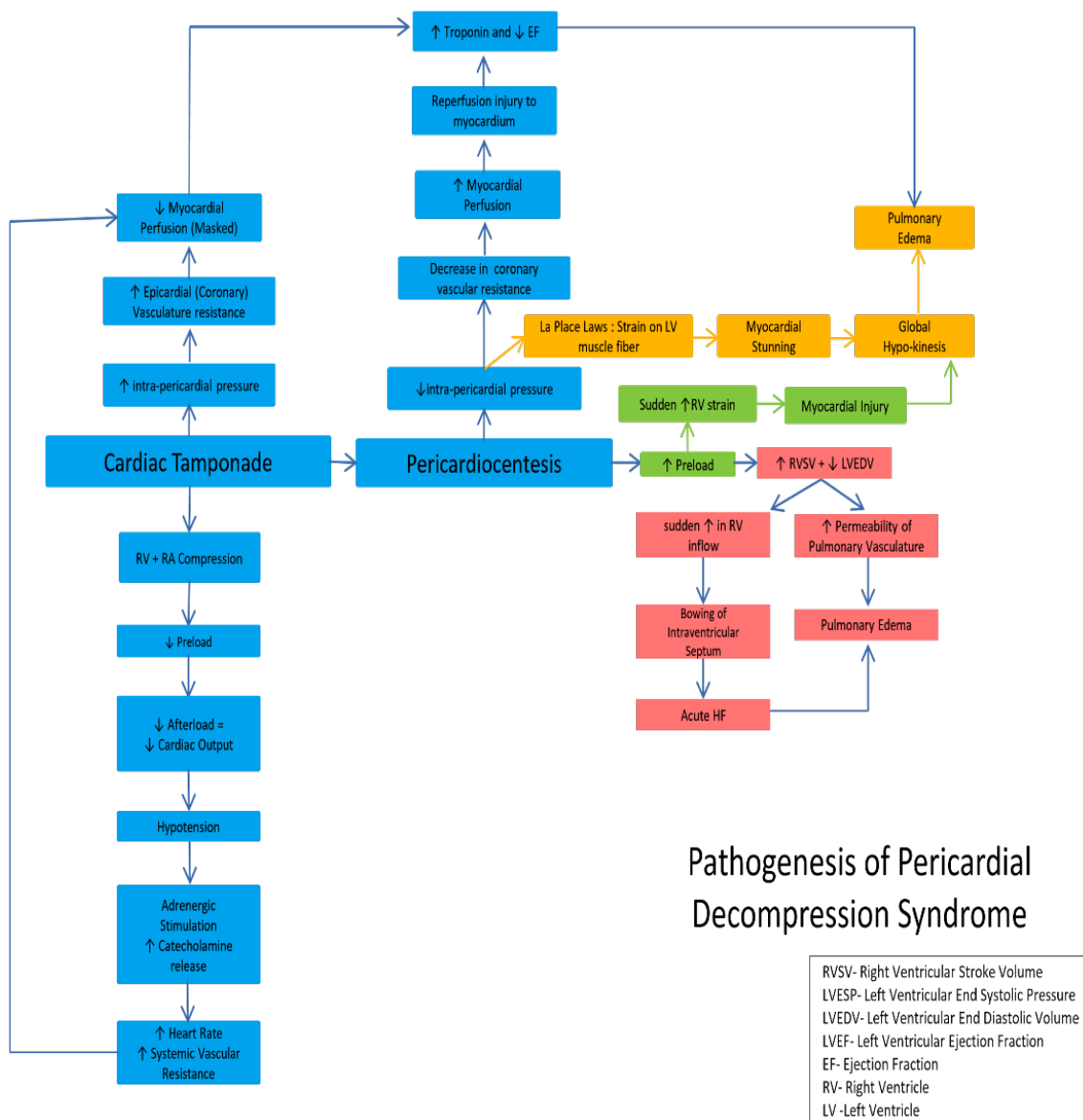


Fig. (1). PDS Flow Chart. (A higher resolution / colour version of this figure is available in the electronic copy of the article).

injury. A total of 16 patients had troponin checked and 56.3% (n=9) had elevated troponin. Once decompression occurs, the ventricular dysfunction is more evident. This change is also evident in this study with the mean pre-procedural EF 56.2 ± 14.4 compared to the mean post-procedural EF 29.6 ± 14.5 .

Overall, and from our review of the literature, it seems that PDS can occur secondary to multiple pathogenic processes (Fig. 1). However, which one predominates is probably related to the patient and effusion characteristics. Future work should concentrate on identifying these patients' specific and effusion specific risk factors.

4.4. Management

The approach towards management has to be multifaceted, given the potentially multifactorial pathogenesis. The treatment goal is to provide supportive care and minimize complications until full recovery, making prevention of this

condition of paramount importance [50]. In our literature search, we found neither guidelines nor recommendations for the prevention or management of PDS. Most authors recommended the gradual removal of fluid from the pericardium [3, 13, 37, 50, 61, 62]. This was not always successful since drainage of 150 ml in 4 hours, which roughly equates to a rate of 37.5 mL/hr, caused hemodynamic instability and biventricular failure [63].

PDS prevention is necessary as its diagnosis carries a high mortality burden [64, 65]. Multiple examples denote that the rate of drainage [66], as well as the total volume drained, has a little role in preventing PDS [30-32]. Imazio *et al.*, recommend the aspiration of fluid to relieve hemodynamic instability; this treatment would be followed by the placement of a long-term drain for further management. Once drainage is confirmed to be less than 30ml/day, the drain can successfully be removed [50].

It is preferable that drainage of pericardial fluid should be attempted in a unit capable of airway management and inotropic support. As per our analysis, n=40 of patients required inotropic support. The majority of the patients who developed PDS had a respiratory failure, which may eventually require intubation. Authors have also reported the use of VA-ECMO, which leads to favorable outcomes [27]. Lango *et al.*, reported high volume hemofiltration as an adjunctive treatment for PDS as well [23].

4.5. Prognosis

PDS is associated with high mortality. The postoperative mortality after subxiphoid pericardiocentesis and pericardiostomy in cancer patients is attributed to low cardiac output state and ranges from 20-24% [67]. As per our analysis, 22.5% (n=14) of patients with PDS expired. The outcome of death is significantly higher in patients who underwent pericardiostomy with a p <0.0001. Patients who survive may recover adequate ventricular function if provided with appropriate supportive treatment. As per our data, a total of 26 patients recovered their ejection fraction after ventricular failure. The recovery period ranged from 1-21 days.

CONCLUSION

Pericardial decompression syndrome is a rare condition with high mortality, and operators performing pericardial drainage should be aware of this complication following pericardiocentesis and/or pericardiostomy of cardiac tamponade. Early recognition and expeditious supportive care are the only therapeutic modalities available for adequate management of this complication. Further research should identify personal and pericardial fluid characteristics associated with this phenomenon.

CONSENT FOR PUBLICATION

Not applicable.

STANDARD OF REPORTING

PRISMA guidelines and methodology were followed in this study.

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CONFLICT OF INTEREST

The authors declare no conflict of interest, financial or otherwise.

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